

CLINICAL LECTURES
ON DISEASES OF
THE LUNGS & THE HEART

J. A. LINDSAY

R.C.P. EDINBURGH LIBRARY

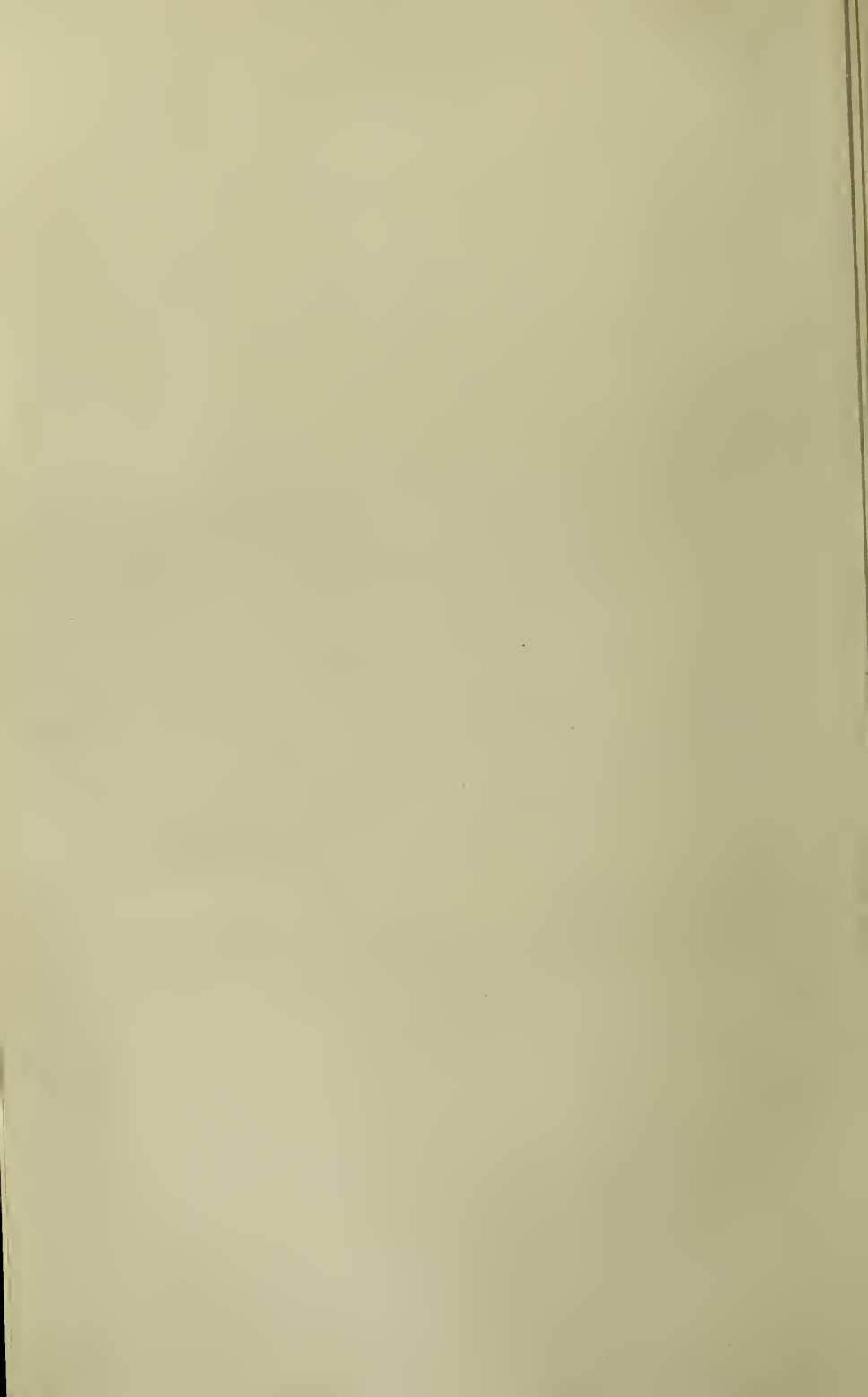


R27706J0236



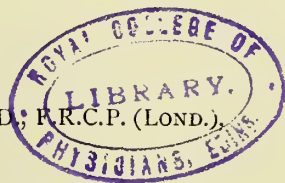
T. 4. 16.

LECTURES
ON
DISEASES OF THE LUNGS AND
THE HEART



LECTURES
CHIEFLY CLINICAL AND PRACTICAL
ON
DISEASES OF THE LUNGS AND
THE HEART

BY
JAMES ALEXANDER LINDSAY, M.D., F.R.C.P. (LOND.),
M.A.

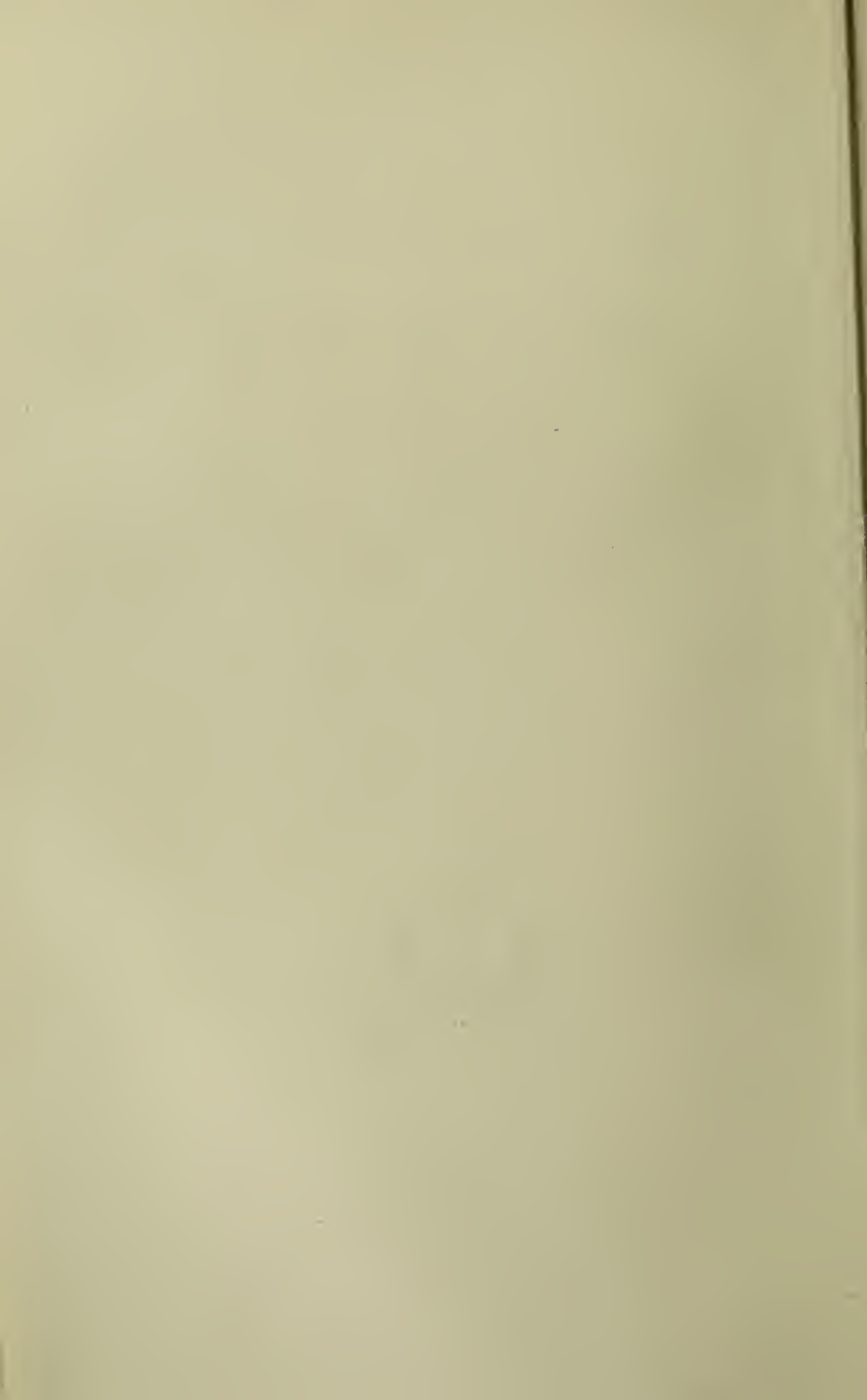


PROFESSOR OF MEDICINE, QUEEN'S COLLEGE, BELFAST
PHYSICIAN TO THE ROYAL VICTORIA HOSPITAL, BELFAST
CONSULTING PHYSICIAN TO THE ULSTER EYE, EAR, AND THROAT HOSPITAL, BELFAST
THE ULSTER HOSPITAL FOR CHILDREN AND WOMEN, BELFAST
AND THE COLERAINE COTTAGE HOSPITAL
EXAMINER IN MEDICINE IN THE ROYAL UNIVERSITY OF IRELAND
LATE PRESIDENT OF THE ULSTER MEDICAL SOCIETY



LONDON
BAILLIÈRE, TINDALL AND COX
HENRIETTA STREET, COVENT GARDEN

1904



P R E F A C E .

THE lectures contained in this volume have been in substance delivered to my clinical classes at the Royal Victoria Hospital during the past fifteen years. They have been somewhat altered in form, with the view of rendering them more suitable for publication, and much new matter has been added.

The lectures, though not systematic in point of form, are intended to raise and discuss most of the problems of diagnosis, prognosis, and treatment which confront the practitioner in the field of thoracic disease. Difficulties of diagnosis have received special attention. The object of the lectures is practical, and the clinical standpoint has been adopted throughout.

Two lectures—viz. I. and II.—are included in this volume which are not directly concerned with diseases of the lungs and the heart, but rather with the theory of clinical observation and the philosophy of medicine in general.

I have freely consulted the extensive literature of the subject—English, American, French, and German—and I am much indebted to the leading authorities in this field

of medicine. I have endeavoured to acknowledge my obligations as fully as possible.

Some of my former pupils have desired to have my teaching in permanent form. To them, therefore, and to my students of to-day I dedicate this volume.

J. A. L.

13, COLLEGE SQUARE EAST,
BELFAST,
January, 1904.

CONTENTS.



LECTURE	PAGE
I. DIAGNOSTIC METHOD	1
II. THE INTERPRETATION OF HISTORY IN DISEASE ...	15
III. PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (A)	35
IV. PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (B)	55
V. PLEURISY	84
VI. THE EARLY DIAGNOSIS OF PULMONARY PHTHISIS	112
VII. CONDITIONS WHICH SIMULATE PULMONARY PHTHISIS	135
VIII. PROGNOSIS IN PULMONARY PHTHISIS	151
IX. THE TREATMENT OF PULMONARY PHTHISIS ...	171
X. THE TREATMENT OF PULMONARY PHTHISIS (<i>con- cluded</i>)	192
XI. THE CAUSES AND MANAGEMENT OF HÆMOPTYSIS	222
XII. SOME OF THE RARER FORMS OF PULMONARY DISEASE	242
XIII. SOME THERAPEUTIC PROBLEMS IN PULMONARY DISEASE	256
XIV. PHYSICAL EXAMINATION OF THE HEART ...	272
XV. PHYSICAL DIAGNOSIS OF CARDIAC DISEASE (CAR- DIAC MURMURS)	293

LECTURE	PAGE
XVI. PHYSICAL EXAMINATION OF THE BLOOD-VESSELS ...	318
XVII. THE STUDY OF CARDIAC SYMPTOMS	340
XVIII. DISORDERS OF THE CARDIAC RHYTHM	358
XIX. SOME DIAGNOSTIC PROBLEMS IN CONNECTION WITH HEART DISEASE	377
XX. PROGNOSIS IN CHRONIC VALVULAR DISEASE OF THE HEART	394
XXI. THE TREATMENT OF VALVULAR DISEASE OF THE HEART	416
INDEX OF AUTHORITIES	439
GENERAL INDEX	441

DISEASES OF THE LUNGS AND HEART.



LECTURE I.

DIAGNOSTIC METHOD.

SUMMARY.

Two methods employed in diagnosis, viz.—

A. The Serial Method.

B. The Direct Method.

Examples of these methods.

A. The Serial Method.

Advantages—

- (a) It secures completeness.
- (b) It minimises the risk of premature, incomplete, or erroneous diagnosis.
- (c) It ensures the detection of complications.
- (d) It involves no assumptions.
- (e) It suits the learner.
- (f) It suits the investigation of obscure cases.

Disadvantages—

- (a) It may be slow and cumbersome.
- (b) It may lead to confusion between recent and old-standing lesions.

B. The Direct Method.

Advantages—

- (a) It is prompt, efficient, and time-saving.
- (b) It is the natural method of experience.

- (c) It is the natural method in simple cases.
- (d) It is applicable to most cases, except the very obscure.

Disadvantages—

- (a) It may encourage premature conclusions.
- (b) It may lead to the neglect of complications.
- (c) It is unsuitable to obscure cases.

Comparison of the two methods.

Examples and cautions.

IN the diagnosis of disease we proceed by one or other of two methods. Having obtained the history and elicited the symptoms, we either—

- (a) Proceed to examine the various organs of the body in definite serial order, and having thus collected the facts, we proceed to draw our inferences and formulate our diagnosis (Serial Method); or
- (b) The history and symptoms having suggested some particular lesion, we seek for that lesion, and, having found it, the diagnosis is established (Direct Method).

As an example of the Serial Method, we may take the following case :—

A patient presents himself, complaining of debility, loss of flesh, impairment of appetite, shortness of breath. Here the indications are obscure, the possible explanations many. We proceed by the Serial Method. We examine in order the superficial parts, the lungs, the heart, the digestive organs, the blood, the urine, and the nervous system. We may find that the symptoms depend on anæmia, dyspepsia, phthisis, heart disease, Bright's disease, organic nerve disease, or some other condition.

As an example of the Direct Method, we may take the following case :—

A patient presents himself, complaining of cough, spitting of blood, loss of weight, and night-sweating. Here the indications, though not decisive, are strongly suggestive

of a certain disease. We proceed by the Direct Method. We examine the lungs, find evidence of tubercular deposit, and thus arrive at a diagnosis.

I propose to subject these two methods to analysis, to point out the range of applicability of each, to consider their respective advantages and disadvantages, and the fallacies to which each method is specially liable.

A. THE SERIAL METHOD.

We may say, generally, regarding this method, that it secures completeness, reduces the risk of premature and incomplete diagnosis, ensures or at least facilitates the detection of complications, is applicable to all cases (though not convenient in all), is the natural method of the learner, is specially suitable in obscure cases and in research work.

On the other hand, the Serial Method may be sometimes slow and cumbersome. It is often obviously unnecessary. If not carefully employed, it may lead to confusion between recent and old lesions.

Advantages of the Serial Method.

That the Serial Method *secures completeness* is obvious. It is also obvious that no other method can do so in equal degree. If we form a hypothetical diagnosis from the history and symptoms, *i.e.* if we proceed by the Direct Method, verify this by attention to physical signs, and then look for the usual complications, there is still a danger that rare complications or co-existing morbid conditions may be overlooked.

The Serial Method *minimizes the risk of premature, incomplete, or erroneous diagnosis*. *Ex hypothesi*, in the employment of this method, we collect all the facts before

proceeding to draw any inference. The mental habit thus cultivated is of great importance.

The Serial Method *ensures the detection of complications*. If every organ be adequately examined, it is clear that no complications will be overlooked.

The Serial Method *involves no assumptions*. Perhaps the most fertile source of error in medical diagnosis, next to hasty and inaccurate observation of facts, is the premature assumption of ill-founded conclusions. The Serial Method proceeds on the basis of no assumptions. Its essential principle is the collection of all the facts before any theory is formulated.

The Serial Method *suits the learner*. It is, in fact, the only method by which the learner can make progress. The beginner in medicine does not possess the general knowledge of disease which renders the Direct Method safe and fruitful to the experienced observer.

The Serial Method *suits the investigation of obscure cases, and is the natural method of research*. Where we are dealing with cases where the history and the symptoms suggest no probable diagnosis, or—what is not less common—suggest various possible diagnoses, it is clear that the only safe plan is to proceed to examine all the organs of the body in definite serial order. In research work completeness is obviously indispensable.

Disadvantages of the Serial Method.

The Serial Method *may be often obviously inapplicable*, *e.g.* in pneumonia, we do not find a minute examination of the nervous system necessary ; in psoriasis, an exhaustive inquiry into the state of lungs or heart would usually be wasted ; in aneurism of the aorta, a detailed investigation of the blood is usually supererogatory.

It is to be observed, however, that the assumption that

the state of certain organs has no bearing upon a certain disease is always open to fallacy. Such an assumption presupposes a complete knowledge of disease, which, in a large proportion of cases, we do not possess. As examples of such errors, we may take the following: A minute examination of the condition of the bones and joints in *tabes dorsalis* might have seemed superfluous until Charcot taught us to recognize the arthropathies and osteopathies of that disease. The state of the pupil might have seemed unlikely to throw any light upon the same disease until Argyll-Robertson taught us its great importance. The state of the thyroid gland might have seemed irrelevant in certain states of debility and mental enfeeblement until Ord divined their relation.

The assumption that in any morbid condition we can safely ignore the state of any organ, though often quite warranted, is always open to fallacy. The probability of such fallacy varies in different cases from almost zero to a serious risk.

The Serial Method *may be slow and cumbersome*. A careful examination of all the organs of the body would not be necessary or convenient in making a diagnosis in a case of, *e.g.*, scarlatina, herpes zoster, chorea, exophthalmic goitre, myxœdema. In such cases—and the list might be easily enlarged—the indications afforded by the history and symptoms are usually of so decided a nature that the question which presents itself for solution is not so much, What disease is the patient suffering from? as, Is this a case of scarlatina, chorea, myxœdema, etc., or, if not, what condition have I before me which simulates one of those diseases?

As an example, however, of a case which may seem clear and yet lead to error if the Serial Method is neglected, we may take a case of facial paralysis. Here the

condition is to a certain extent obvious. The exploration of the heart, the lungs, the urine, etc., may seem superfluous. Yet facial paralysis may be either of peripheral or centric origin; if of the latter, the morbid condition in the brain may depend on vascular disease, this in its turn upon atheroma, the atheroma may be related to syphilis or to renal disease. These conditions may only reveal themselves upon a careful application of the Serial Method. Thus it is evident that even in a case of apparent simplicity danger may lurk in the application of the Direct Method.

The Serial Method *may lead to confusion between recent and old-standing lesions*. This must not be regarded as an essential vice of the method, but rather as apt to arise from its illegitimate application. As an example of this danger, we may take the following instance: A patient presents himself with some obscure nervous symptoms. A serial examination is instituted and mitral disease is discovered. The symptoms are attributed to this cause, whereas in reality they are due to commencing disseminated sclerosis or to some form of toxæmia. This may seem a somewhat gross case of hasty and inadequate diagnosis, but parallel errors are not uncommon. It is evident, however, that such an erroneous diagnosis involves an illegitimate employment of the Serial Method. In fact, the case in point involves two errors—first, attributing to mitral disease symptoms not likely to depend on that condition; and, secondly, overlooking other features of the case which might have suggested the true diagnosis.

B. THE DIRECT METHOD.

The Direct Method consists, as already stated, in forming a hypothetical diagnosis from an analysis of the

history and the symptoms, and then confirming or correcting that diagnosis by an investigation of the physical signs.

Advantages of the Direct Method.

The Direct Method is *prompt, efficient, and time-saving*. The history and symptoms having strongly suggested the existence of a certain lesion, we look for that lesion, and having found it, the diagnosis is established. In skilful and experienced hands nothing can be simpler or more efficient. Time is saved; the attention is not distracted to irrelevant issues.

The Direct Method *is the natural method of experience*. The physician of large experience draws his conclusions from the history and the symptoms with more or less confidence, and looks to physical signs to corroborate, correct, or exclude his hypothetical diagnosis. His knowledge of disease enables him to form a probable theory as to the nature of the case before him, and saves him from barren and unlikely guesses. The unskilled observer of small experience obviously applies the Direct Method under great disadvantages. The history and symptoms may either suggest to him an erroneous diagnosis or no diagnosis at all.

The Direct Method *is the natural method in simple cases*. Many cases of disease are obvious to the trained observer. A case of psoriasis, or Graves's disease, or acromegaly may sometimes be recognized at a glance, and while a minute exploration of the various organs may throw light on prognosis or modify treatment, it adds nothing to the diagnosis.

The Direct Method *is applicable to most cases*—excluding those which are very obscure. As we listen to a patient's story of his history and symptoms our minds naturally

tend to form a theory or theories as to the nature of the case. In proportion to our knowledge and experience, do we feel confidence in the correctness of the hypothesis thus arrived at. Our hypothesis may possess varying degrees of probability. It may either point almost irresistibly to a certain lesion, or may suggest two lesions to the exclusion of all others, or may present three or more alternatives.

Disadvantages of the Direct Method.

The Direct Method may *encourage a premature conclusion*. The history and symptoms may suggest a diagnosis true as far as it goes, but incomplete and misleading.

A patient may complain of cough, expectoration, and dyspnoea, a diagnosis of bronchitis is conjectured, and physical signs may seem to give it support. But the really substantive condition may be typhoid fever or tuberculosis, and the diagnosis of bronchitis—in a sense correct, so far as it goes—may be wholly inadequate and erroneous. Or a patient may complain of loss of colour, breathlessness, and impaired digestion. By application of the Direct Method anæmia is looked for and found; but the anæmia may be symptomatic of renal disease, or aortic regurgitation, or tuberculosis, or cancer, and may be quite misleading if accepted as a substantive diagnosis.

The Direct Method may *lead to the neglect of complications*. If the observer, having correctly inferred from the history and the symptoms the existence of a certain lesion, and having proved by physical examination the presence of that lesion, is either ignorant of the probable complications or careless in his search for them, it is evident that error will arise.

The Direct Method is *unsuitable to obscure cases*. If the history and the symptoms make no definite suggestion, it is evident that the Serial, and not the Direct, Method is required.

The Direct Method is *unsuitable for the beginner*. It will be evident, on a little reflection, that the Direct Method is applicable and valuable just in proportion to the knowledge and experience of the observer. To the experienced observer the history and the symptoms suggest, in a large proportion of cases, their true inference. To the novice they may suggest either an erroneous inference or no inference at all.

As an example of the different value of the Direct Method in skilled and unskilled hands, I may cite the following case: A patient consulted a doctor for persistent irritating cough. The lungs, the heart, and other organs were examined without any physical signs being discovered. Treatment having proved unavailing, a consultant was called in; and he, on hearing the history and the symptoms, looked for and found an elongated uvula.

The Direct Method must always be regarded as incomplete until we have answered the question, Does the diagnosis fully account for the history and the symptoms? The neglect of this step is one of the most fertile sources of error in medical practice.

As an example of this error, I give the following case from my experience as an examiner:—

A patient was admitted to hospital complaining of cough, breathlessness, and pain in the chest. A good deal of general bronchial catarrh existed. A thoracic aneurism, of which the signs were slight but decisive, was discovered, and this was, no doubt, the cause of the patient's symptoms. A candidate to whom the case was assigned at

his examination, gave a diagnosis of "chronic bronchitis." He was asked if his diagnosis fully accounted for the history and the symptoms (persistent, deep-seated thoracic pain and brassy cough were prominent features of the case). He thought it did. He had noted the presence of pain, but did not think it important. The possibility of aneurism had not occurred to him.

The process of reasoning in the candidate's mind was no doubt as follows: I find a patient complaining of cough, breathlessness, etc. On examination I find the physical signs of bronchial catarrh. I infer that the case is one of chronic bronchitis. He forgot to put to himself the question, Does my diagnosis fully explain the history and the symptoms?

As an example of the correct application of this rule, I give the following example:—

A gentleman, aged about fifty, was taken ill with a rigor and marked pyrexia (104°). He was attended by his family doctor, the symptoms suggesting pneumonia, but the physical signs being obscure. There was marked dyspnœa, a high and rather irregular pyrexia, a frequent weak pulse, much prostration, and a dry, cracked tongue. On the ninth day he was seen by a consultant, who found a small patch of crepitation at the base of the left lung. On inquiry into the mode of onset, it appeared that severe frontal headache had been a feature of the initial stage. There were no abdominal symptoms and no rash. The question being put, Does a small patch of crepitation at the base of the left lung account for the history and the symptoms? the answer was, No; it does not account for the initial frontal headache, the severity of the symptoms, the amount of prostration, the state of the tongue. The suggestion was hazarded that the case was one of typhoid fever with pulmonary complications. And so it proved to

be. Severe intestinal hæmorrhage set in a few days later, and the patient sank.

This case illustrates several important points. It shows the necessity of putting the question, Does the diagnosis fully explain the history and the symptoms? It also illustrates the possibility that an important complication may easily and naturally be mistaken for the substantive disease.

Upon a comparison of the Serial Method and the Direct Method, we may observe—

- (a) That the Serial Method is *safer*, the Direct Method *easier*.
- (b) That the Serial Method *ensures completeness*, the Direct Method *favours promptness*, of diagnosis.
- (c) That the Serial Method is the safeguard of the young practitioner, the Direct Method is the resource of the experienced physician.
- (d) That the Serial Method is to be preferred in dealing with obscure, the Direct Method in investigating simple, cases.
- (e) That a combination of the two methods may be preferable to the application of either method taken singly.

The Combined Method is specially suitable to cases which have the appearance of simplicity, but which become more complex the more they are investigated. Thus the history and the symptoms frequently suggest a diagnosis which, on carefully collecting the physical signs, turns out to be untenable. A patient complains of cough, wasting, feverishness, and night-sweating. A diagnosis of phthisis is naturally inferred. But, on examination, the physical signs do not support this diagnosis, and no bacilli are found in the sputum. How do we proceed? Either by inquiring what condition, or conditions, other than

tuberculosis, might explain the history and the symptoms (*e.g.* syphilis, carcinoma), *i.e.* by a further application of the Direct Method, or by falling back upon the Serial Method, which may reveal to us such helpful facts as the presence of malignant disease in some other organ, or cicatrices on the penis, or secondary degenerations of the nervous system.

The Direct Method may mislead us by suggesting as the cause of a condition what is really a joint effect of that condition.

Let us take an example.

A patient complains of anæmia, debility, dyspepsia. By the application of the Direct Method we conjecture that renal disease is the cause, and on examining the urine, albumen is found to be present. If we conclude without further inquiry that albuminuria is the cause of the patient's symptoms we may fall into serious error. The anæmia and debility on the one hand, and the albuminuria on the other, may both be effects of the real cause, *e.g.* amyloid degeneration, depending on bone disease, syphilis or phthisis.

Or, a young girl complains of weakness in her legs, tremors, emotional disturbance. We suspect hysteria (by an application of the Direct Method), and on examination proof of hysteria is elicited. But the symptoms complained of and the hysterical phenomena may be joint effects of a common cause, *e.g.* nervous heredity, worry, shock.

As an example of the erroneous application of the Direct Method, I take the following case from my own experience:—

Miss X., aged 35 years, who had previously enjoyed good health, has been nervous, excitable, or depressed for a year or more. Alleged cause, a disappointment in love. She is odd in manner, and often breaks out in causeless

laughter. She often lets objects fall from her hands, and occasionally has some difficulty in feeding herself. She complains of weakness in her legs, and is easily fatigued. There is slight tremor of the hands and arms, increased on exertion. The knee jerks are much increased on both sides. There is no ankle clonus. There are no trophic changes. The patient is "hysterical" in manner. There is no nystagmus. There is no "scanning" speech.

It was natural in this case to apply the Direct Method. The history and the symptoms suggested hysteria, and on a first examination there was some ground for affirming, and no ground for definitely excluding, this diagnosis. As a matter of fact, hysteria was diagnosed. But in a few months clear evidence of commencing disseminated sclerosis was forthcoming.

How could this error have been avoided?

The Combined Method would in this case have been the most suitable, the case being only superficially obvious and really obscure. The Direct Method suggested hysteria, but on inquiry the evidence for this diagnosis, though amounting to a certain probability, was not conclusive. The Serial Method should, then, have been rigorously applied, and the thorough examination of the nervous system involved in this application might have resulted in the discovery of some point or points decisive of organic nerve disease. If so, hysteria would be excluded as a complete diagnosis. If such point or points were not discovered, the Serial Method would have presented the facts of the case in the following form: I find a train of nervous symptoms suggestive of hysteria, but consistent, also, with the diagnosis of commencing organic nerve disease. Of definite proof of the latter I can find no evidence. The case, therefore, remains open, and time will be required to clear it up.

Both the Serial and the Direct Method are open to the objection that they may lead to no conclusion.

I find symptoms *a, b, c, d*, and physical signs i., ii., iii., iv.

Having collected these by the Serial Method, I inquire, What conclusion do these facts warrant? The answer is, No conclusion.

Or, I elicit the symptoms and the physical signs, and attempt to apply the Direct Method by asking, What organ is probably at fault? The answer is, It is impossible to say.

In such circumstances we do not conclude that we are dealing with a new disease—which we know to be highly improbable—but we infer that either we have overlooked something, or else that the disease is not sufficiently developed to render diagnosis possible.

LECTURE II.

THE INTERPRETATION OF HISTORY IN DISEASE

SUMMARY.

History is of two kinds—

- A. Family.
- B. Personal.

A. Family History.

Family diseases, inherited proclivity to infection, family idiosyncrasies, misleading histories.

B. Personal History.

Preliminary questions, importance of getting patient's own story danger of leading questions, their occasional utility, difficulty of estimating the duration of disease, importance of a correct account of initial symptoms, illustrative cases.

Errors of patients in giving a history of their diseases :—

- (a) They fail to note, or erroneously describe, existing symptoms.
- (b) They fail to note the first beginnings of disease.
- (c) They assign erroneous causes to disease.
- (d) They knowingly withhold the true cause or causes of disease.
- (e) They give an incorrect account of habits.
- (f) They place a false emphasis on certain symptoms.

Illustrations and cautions.

MEDICAL history is of two kinds: A. Family; B. Personal

A. FAMILY HISTORY.

We seek information—

- (a) As regards family diseases ;
- (b) As regards inherited proclivity to infection ;
- (c) As regards family idiosyncrasies.

(a) The following diseases are known to be hereditary, viz. gout, rheumatism, syphilis, cancer, diabetes, asthma, heart disease, chlorosis, hæmophilia, ichthyosis, alcoholism, neurasthenia, hysteria, chorea, insanity, epilepsy, migraine, colour-blindness, apoplexy, pseudo-hypertrophic paralysis, idiopathic muscular atrophy, Friedreich's disease.

It is not to be supposed that this catalogue exhausts the list of inheritable diseases. Inheritance affects all the tissues and organs of the body, and its influence upon disease may be potent, although not obvious. The individual does not inherit in most cases the peculiarities of his parents, but rather the general characters of his stock on both sides. In some cases inheritance is definitely from the paternal or the maternal side. Nothing is clearly known as to the laws which determine inheritance from one side or the other. Prepotency, which is so obvious in the case of the lower animals, is also important in the human species.

In certain cases inheritance is to the males of the stock through females, who themselves escape. The most typical case is hæmophilia. This law is also seen in pseudo-hypertrophic paralysis, colour-blindness, gout, and some other conditions.

The fact of inheritance may be obscured because a morbid tendency assumes different forms in successive generations. This is seen particularly in nervous diseases. Alcoholism in one generation may be represented by insanity in the next. The child of a neurotic mother is the victim of epilepsy or migraine. What is inherited is congenital weakness of nervous tissue.

The same law holds as regards the vascular system. Heart disease, aneurism, premature senility of the arteries, apoplexy, renal disease appear in successive generations,

the underlying fact being that the individual inherits the characteristics of his stock *quoad* his blood-vessels.

(b) We seek information as regards *inherited proclivity to infection*. Tuberculosis is here the leading case. That this disease runs in families is one of the most familiar and certain facts in medicine. But tuberculosis is not hereditary in the strict sense of the term. The child is not born tubercular, nor does it at birth possess the germs of the disease,* nor is it inevitable that the child of the most tubercular stock must necessarily develop tuberculosis. What is inherited is a proclivity to infection by the bacillus of tuberculosis.

A similar law may be more or less observed in the case of some of the exanthemata. In certain families every member suffers from scarlatina. In other families every member, in spite of exposure to contagion, escapes. We must assume a congenital proclivity to, or immunity from, infection.

(c) We seek information regarding *family idiosyncrasies*. In this connection the original number of members of the patient's family, the number of surviving members, and the age at time of death of the deceased members, are facts of great importance. Amongst hospital patients accurate details as regards the causes of death of patient's relatives are often difficult to obtain. Certain rules—to be interpreted with some reserve—will help us here. These rules are as follows:—

1. Several deaths in early childhood amongst members of the same family suggest as a probable cause one of the specific fevers, *e.g.* measles, whooping cough, scarlatina, diphtheria.

2. Several deaths in adolescence or early adult life amongst members of the same family suggest as a probable cause, tuberculosis.

* I do not accept Baumgarten's theory on this subject.

3. Several deaths at middle life amongst members of the same family suggest as a probable cause premature vascular degeneration, *e.g.* heart disease, aneurism, apoplexy.

Patients often mislead us, wilfully or otherwise, regarding the facts of family history.

Tuberculosis is concealed under such expressions as "died from the effects of a chill," "blood from the throat," "disease of the bowels," "water on the brain." Cancer is concealed under some vague story of a doubtful tumour, or of "death from the effects of an operation." Insanity is concealed under such expressions as "brain disease following a confinement." Alcoholism, delirium tremens, and syphilis are explained away or glossed over more or less plausibly. In such cases the observer will do well to bear in mind the really probable alternatives, and according to the facts of the case ask himself the question, Do these vague expressions really cover a history of tuberculosis, or cancer, or syphilis, or insanity, or alcoholism? If such problems are concretely present to our minds, confirmation of our suspicions will often be obtainable by indirect evidence.

Certain family peculiarities may prove to be of importance from the medical point of view. Amongst such facts may be enumerated the following: abnormalities as regards weight, diet, average duration of sleep, loss of teeth, capacity for enduring fatigue, effects of cold baths.

B. PERSONAL HISTORY.

The ideal would be that the patient should give a complete and accurate account of the origin of his illness and of his symptoms, in the order of time, without inferences. The average patient is incapable of fulfilling these conditions. He fails to note the first significant

departures from health, he overlooks or misdescribes existing symptoms, he assigns erroneous causes, he mixes up fact and inference.

In most cases it is well to begin by putting the following three elementary questions:—

1. What do you complain of?
2. How long are you ill?
3. How did your illness begin?

1. It is of the first importance to get the patient to state clearly and fully what he complains of, what he himself feels to be amiss, what he seeks advice and assistance for. This may seem an obvious rule, but it is often forgotten, and the neglect of it is a fertile source of error. The patient constantly volunteers some ready-made diagnosis or informs us that some other adviser has said this or that. It requires a real effort of mind—especially on the part of the uneducated patient—for him to grasp the fact that we want his own story, a plain, unvarnished tale of how he actually feels, wherein in his own judgment his condition departs from, or falls short of, health. If his attention is kept firmly fixed to this point, we get information of greater or less value, according to the intelligence of the patient, but always of some value.

If we do not make the patient's actual symptoms our *point de départ* there is always the possibility of serious error. If, after a few cursory questions, the physician, full of the conviction that physical investigation is the *unum necessarium*, proceeds to examine, there is always the danger that he may discover some chronic lesion which is not the source of the patient's symptoms, and may thereby be led into a wrong track. A patient seeking relief from a trigeminal neuralgia has been known—incredible as it may appear—to leave the outpatient department of a hospital with a prescription

directed towards a mitral murmur which had no relation to his actual symptoms. A young woman with commencing disseminated sclerosis may be put under treatment for ovaritis or anæmia because the physician looks for signs and is not sufficiently solicitous about symptoms. An obvious bronchitis is treated while a thoracic tumour is overlooked, because the observer did not take sufficient pains to note that deep-seated pain was one of the prominent features.

In all such cases there is a neglect of the rule laid down in connection with our discussion of the Direct Method of diagnosis, viz. that we must always put to ourselves the question, Does the diagnosis at which I have arrived fully explain the patient's history and symptoms?

It is not to be expected that patients, untrained in observation and ignorant of disease, will give a satisfactory account of their symptoms. Often enough the account given is meagre and inaccurate. On the other hand, when symptoms have become sufficiently important to require medical advice, they have in many cases come to bulk largely in the patient's mind; they are matters of vital moment to him, and many patients are very self-observant. In most cases it is well to get the patient to tell his own story, and to avoid putting leading questions. When the patient has given what is often a rambling and confused story, the physician may often do well to sum up its purport in some such way as this: You complain of such and such symptoms. Have you anything to add to this account before I proceed to examine you? Often enough the postscript to the patient's story contains the real marrow of the case.

There is one case where putting leading questions is not only permissible, but even of considerable diagnostic value—I mean where suggestion plays a large part. In cases of

neurasthenia, hysteria, and allied states, the patient often responds eagerly when new symptoms are suggested. Having complained only of pain in the head, the patient responds affirmatively when pain in the back, the lower limbs, or the ovarian region is inquired for. Having complained of buzzing in the ears, or black specks before the eyes, the patient will sometimes invent a long train of auditory and visual phenomena if these are suggested by leading questions. This peculiarity—this responsiveness to suggestion—may be of considerable importance in differentiating between such conditions as the following :—

- (a) Neurasthenia and commencing tabes dorsalis, or general paralysis of the insane.
- (b) Hysteria and commencing disseminated sclerosis.
- (c) Neurasthenic headache and brain tumour.
- (d) Nervous palpitation and organic heart disease.

We must remember that responsiveness to suggestion makes functional disorder probable, but in no way excludes the possibility of organic disease. In certain conditions, *e.g.* diseases of the heart and of the central nervous system, the symptom-complex is often composed partly of symptoms of organic disease and partly of symptoms due to functional complications. Such cases are specially apt to lead to error and confusion in diagnosis. The most usual error is that the functional complication overshadows the organic mischief, and is regarded as the sole existing condition. But the opposite error is not unknown. In analysing the symptom-complex of a case, *e.g.* of organic heart disease, the observer, having a definite structural lesion before his mind, does not always stop to reflect that some of the symptoms may be functional in character. The palpitation of mitral regurgitation may be, and usually is, of organic causation. But there is no reason why it may not sometimes be due to functional causes.

2. The second indispensable preliminary question is, How long are you ill?

In recent and acute cases the answer to this question will often be accurate, but in chronic cases of insidious onset the largest errors are possible. Members of the artisan and labouring classes, unaccustomed to pay minute attention to their health and habituated to daily labour, so long as labour is possible, commonly underestimate the duration of their illness. They date the onset not so much from the first advent of symptoms as from the time when symptoms became too obtrusive to be any longer ignored. The cultivated and leisured classes, on the contrary, accustomed to care and watchfulness in matters relating to health, usually note early symptoms of illness, and fix their date with approximate accuracy. In dealing with the former class, it is always well to find out upon what date illness first prevented daily labour. That forms a landmark of importance which is usually remembered.

3. The third indispensable preliminary question is, How did your illness begin?

Few things are more helpful in the diagnosis of obscure cases of disease than an accurate account of initial symptoms; few things are more difficult to obtain. Where physical signs fail us, an accurate analysis of symptoms—and, above all, of initial symptoms—is our main resource.

The cases in which an accurate account of initial symptoms serves to lighten the labour of diagnosis and assist in excluding certain alternatives are very numerous. Let us take a few type cases, fully recognizing that the evidence thus obtained is seldom conclusive, simply that it is invaluable for its suggestiveness.

(a) A case is seen for the first time at the end of the third week of illness, the principal points being continued pyrexia and the general symptoms of the febrile state.

Excluding conditions which would be more or less obvious, the alternatives here will probably be—

1. Typhoid fever.
2. Tuberculosis.
3. Pus.

The initial symptoms, if procurable, would here possess high differential value. The first week of typhoid fever, with its persistent frontal headache, characteristic pyrexia, slight chills, increasing debility, and slight bronchial catarrh, has not much resemblance to the early days of acute tuberculosis or deep-seated abscess. Later on these conditions are much more difficult to differentiate.

(b) An obscure nerve case of some months' duration is seen for the first time, and the diagnosis appears to lie between *tabes dorsalis* and general paralysis of the insane. The early symptoms—in the former case of pains, *paræsthesiæ*, ocular changes; in the latter of mental confusion, excitement, fits—would, if correctly observed, go far to establish the diagnosis.

(c) A child is seen on the third or the fourth day of illness, the symptoms being redness of the fauces, a doubtful blush on the chest, pyrexia, and rapid pulse. The diagnosis of scarlatina will be much facilitated if it can be shown that the illness set in suddenly, without obvious cause, and that vomiting was one of the earliest symptoms.

We shall do well to spare no pains in getting as accurate an account as we can, whether from the patient or his friends, of the earliest symptoms of disease. A close scrutiny of such symptoms will often give us the correct clue in the most obscure class of diseases, where physical signs are absent or inconclusive.

I pass on to consider the errors most usually committed by patients in giving us an account of their ailments. These errors have their root either in simple incapacity for

correct observation, in carelessness or inattention, in prejudices regarding medical matters, in timidity or modesty, in deliberate intention to deceive. All these factors will at various times claim our attention.

The most usual errors are the following:—

- (a) Patients fail to note or misdescribe existing symptoms.
- (b) Patients fail to note the first beginnings of disease.
- (c) Patients assign erroneous causes to disease.
- (d) Patients knowingly withhold the real cause.
- (e) Patients give an incorrect account of habits.
- (f) Patients place a false emphasis on certain symptoms.

Of (a) and (b) we have already spoken.

The assignment, in good faith, of erroneous causes of disease is an event of frequent occurrence. In such cases ignorance, prejudice, emotion, tradition play a large part. We shall consider some of the most frequent cases.

Patients *assign many diseases to the effects of chill*. This history is to be received with caution, but not necessarily with incredulity. Chill, understood in the widest sense, is an important factor in the causation of disease. A history of chill is, however, often misleading. In the mouth of a patient it often means nothing more than the feeling of chilliness which is so often one of the earliest premonitory symptoms of febrile disease, and which is not the cause, but the effect, of disease. On the other hand, chill is a potential cause, whether a predisposing or an exciting cause, of a large group of diseases, *e.g.* rheumatism, bronchitis, pneumonia, pleurisy, laryngitis, gastritis, appendicitis, jaundice, Bright's disease, neuritis, myelitis.

To discuss the precise mode in which "chill" operates in these various diseases would lead us far. It may act by "lowering vital resistance"—as the phrase is—and so

render the patient more susceptible of bacterial invasion, as, probably, in pneumonia; or by direct irritative effect, as in laryngitis; or reflexly through the nervous system disturbing the secretions and causing the retention of morbid products in the system, as, possibly, in rheumatism; or in other ways which are more or less theoretical, and need not at present detain us. Our present point is rather to consider how we may distinguish a true history of chill from a false one, recognizing that both are common. We may often get assistance from a minute inquiry into the nature of the chill, whether some mere vague fact of exposure, or a history of a definite wetting, lying on damp grass, or the like. It is important to discover whether the alleged chill was a normal or an abnormal event in the patient's life. A patient of the artisan class often assigns his illness to a wetting received on a particular date, although his avocations may have involved numerous previous wettings without ill effect. Why twenty wettings should have been harmlessly incurred, while the twenty-first precipitates an attack of pneumonia or of pleurisy, does not strike him as a fact requiring explanation. To us it is, of course, the point of real interest.

The precise interval between the alleged chill and the first advent of symptoms may be a fact of importance, though definite rules can hardly be formulated. A chill, if the true causal factor, may be expected to act promptly. It is a matter of hours or days, and not many days. Sometimes patients give a history which is obviously erroneous. An attack of pneumonia or of pleurisy is attributed to a chill which took place one, two, or three weeks before the actual invasion.

Patients often *erroneously attribute many morbid conditions to the effects of accident*. This is sometimes a point of great medico-legal importance. The practice of insuring

against accident serves to multiply such cases, and this is quite natural, and need excite no surprise. The play of self-interest is constant, and its relation to conscious imposture on the one hand and to self-deception on the other is close. The surgical bearings of this question do not fall within my province. Very important, however, to the physician are those cases where accident is blamed as the cause of organic nerve lesions. Few cases in practice require nicer discrimination than these. An unguarded word from the doctor may be the foundation for an ill-founded action at law, while a little judicious discouragement will stifle a frivolous case at the outset. Let us take a concrete example. An individual sustains a fall from a vehicle, or a bicycle, or is present in a railway accident, or is the victim of an assault, and in the course of weeks or months develops organic disease of the brain or cord. What considerations will help us to determine whether there is, or is not, a causal link between the accident and the disease? The question is often much less simple than it at first sight appears. The first point to be determined is, Was the individual in good health at the time of and before the accident? If the answer to this question is in the affirmative, and no other probable cause for the existing disease can be discovered, a certain presumption—not always a strong one—will arise in favour of the lesion and the accident being causally related. If the answer to this question is in the negative, *i.e.* if the individual was ailing at the time of the accident, the problem for solution will be, Does the antecedent morbid condition sufficiently account for the existing lesions without assuming any effect from the accident? Or—and this point is one of great importance—might the pre-existing morbid condition have been aggravated and stimulated to activity by the accident? This last case is not uncommon, and possesses

much medico-legal interest. A nervous disease may exist, but the symptoms may be almost, or even altogether, in abeyance until some form of shock or accident brings them into prominence.

When nervous disease follows some form of shock there are, then, the following alternatives :—

- (a) The shock may be the cause of the lesion.
- (b) The shock may have accelerated the development of a lesion already impending.
- (c) The shock may have brought into prominence symptoms previously latent.
- (d) The relation of the shock to the lesion may have been wholly fortuitous.

The differentiation of these alternatives may involve great difficulties. As an example of the peculiar difficulties which sometimes beset these cases, I cite the following example from my own experience :—

I. M., aged 58, engine-driver, was admitted under my care into the Royal Victoria Hospital, on April 15, 1901, suffering from right hemiplegia.

The history was that the patient was in his usual good health until the previous February 1st. On that day he was in charge of a train, and made a mistake in reading the signals, with the result that his train narrowly escaped a collision with a train coming from the opposite direction, the line being a single one. The trains were pulled up when within a few yards of each other, and no accident took place. No explanation of his failure to read the signals was suggested. He was much shocked, and on arriving home his wife noticed that he could not use his right arm as well as usual, and that his mouth was drawn to the *right* side. He had some difficulty in chewing. He went to bed, and in a few days the symptoms seemed to pass off. On February 6th he made an attempt to resume

work, but had to come home again, as he "felt giddy," and "saw flashes of light and heard noises in his ears." There was no fit, and he did not become unconscious. He remained in a weak state, and on March 3rd he lost the power of walking and took to his bed. He became completely paralyzed on the right side, and began to pass his urine and fæces involuntarily. He was admitted to hospital on April 15th.

On admission, he was found to be suffering from right hemiplegia. Slight power of movement remained in the right arm and the right leg, sensation was somewhat deficient on the right side, the superficial and deep reflexes were not much affected, and there was no ankle clonus. There was some muscular wasting. The patient was dull and stupid. He answered questions slowly, and seemed apathetic to what was going on around him, but recognized his wife when she visited him. He passed his motions involuntarily. The pulse was eighty, full, regular, and of good tension. The sounds of the heart were clear and free from murmur. The arteries were hard and tortuous. The temperature was normal. There was no albumen in the urine. The bowels were confined. The patient's condition gradually became worse. He grew more stupid, and the paralysis deepened. On May 8th he became unconscious, and on May 19th he died. The diagnosis was hemiplegia, depending, probably, on cerebral hæmorrhage.

The patient's wife took an action against the railway company under the Employers' Liability Act. The point at issue was, Was the shock sustained by the patient upon February 1st the sole cause, or a contributory cause, of his illness and death? There were four possible views of the case, viz.—

(1) That the shock caused cerebral hæmorrhage.

- (2) That the shock precipitated an attack of cerebral hæmorrhage which was impending. The patient's age and the state of his arteries were important in this connection.
- (3) That the relation of the shock to the occurrence of cerebral hæmorrhage was purely fortuitous.
- (4) That the attack of cerebral hæmorrhage in reality preceded the shock, and was the cause of the patient's failure to read the signals correctly.

It will be evident, I think, that to decide between these alternatives was not altogether easy. Fortunately, the duties of the medical witnesses were rendered easy by the course which the case took. The judge ruled that, inasmuch as there had been no actual accident, the case did not come within the scope of the Employers' Liability Act, and that hence an action could not lie. My own judgment inclined to the fourth alternative, but the case is interesting as showing that a medico-legal problem, at first sight apparently simple, may on closer examination be found to involve almost insuperable difficulties.

Problems of the above type are a constant source of trouble in the law courts. The medical witness may find it impossible to say "yes" or "no" to a question which seems a plain and simple one to the legal mind.

Patients often *assign an erroneous importance, or want of importance, to certain personal or family peculiarities*. A headache which may portend an on-coming glioma, or a persistent cough really due to incipient phthisis, may be put aside as unimportant because the patient assures us "that he has always been subject to headaches," or "that he has coughed since he was a child." Or a suspiciously low weight is ignored because the patient tells us "that all his people are thin."

Statements of this kind should always be received with

caution. They may be true and relevant. Not rarely they are untrue and misleading.

Patients often *knowingly withhold the true cause, or give an erroneous account of habits*. These two points, which are often closely related, may be conveniently considered together.

As to the former, patients who consult a doctor commonly do so under the wholesome conviction that it is their own interest to tell the truth, and they know that their confidence will be respected. But the truth is not always told. Patients sometimes seek advice for nervous troubles and give us a full account of every presumable cause except the true one—that being perfectly well known to the patient, but something which he wishes to conceal, *e.g.* an attack of syphilis, domestic unhappiness, losses on the Stock Exchange. Needless to say, such cases require delicate handling. The physician must satisfy himself, if possible, of the true state of the case, but it is not always well for him to indicate that he has correctly fathomed it. In such cases the patient sometimes fully recognizes that his secret is guessed, and will accept treatment accordingly, while he is grateful to the physician for not uncovering a hidden wound.

Among the facts which are often withheld are the following: syphilis, gonorrhœa, masturbation, alcoholism, the morphine or cocaine habit, excess in eating, financial losses, domestic worries.

In the case of male patients it is often best to press for the truth as regards sexual diseases or irregularities, though even in such circumstances it may be well at times to exercise some reserve. In the case of females such facts must be inquired into with much caution. Sometimes the truth may be elicited by indirect questions.

Errors as regards eating — errors of excess, less

frequently errors of defect—are among the most frequent causes of disease, and are not always easy to prove. In cases of dyspepsia and disordered nutrition, the practitioner will do well to keep constantly before his mind the simple question, Does this patient habitually eat too much or too little? The true state of the case may not be easy to determine. The free eater may maintain that he practises great moderation, while the underfed may insist that he takes a sufficiency. Exact details should be pressed for, if necessary. It may be laid down as a broad rule that in atonic dyspepsia the patient commonly eats too little, while gastritis is often the result of over-feeding; in hyperchloxydia free eating is common, while in neurotic dyspepsia the appetite and consumption of food are variable.

Errors as regards the character of the food can usually be ascertained without much difficulty. Surprising facts in this connection will sometimes come under the observation of the practitioner. A patient of mine who was advised to make a considerable use of fish in his dietary was found afterwards to have purchased a large salmon and to have lived exclusively on it for some days!

Excess in the use of alcohol is, of course, constantly concealed. The tippler's standard of truth is a low one. Nothing is commoner than for such persons to affirm in the most solemn manner that their daily allowance of alcohol does not exceed a certain definite and moderate maximum, the true facts being otherwise. Where concealed drinking is suspected, the practitioner may get assistance from attention to the following facts—

- (a) The state of the tongue (tremulous and furred) and the breath (foul).
- (b) Morning sickness or vomiting.
- (c) An unduly frequent pulse.

- (d) An air of disorder, nervousness, and carelessness about dress. Some alcoholic cases are, however, watchful about their dress and manner.

The combination of these points is very characteristic. Some of them may be absent.

The following case from my own experience is instructive:—

C. D., aged 47, a master butcher by trade, applied to be examined for life assurance. He gave a history of "never having ailed a day," his appearance was robust, his manner was quiet and natural. All the organs were found healthy. The urine was normal. There was slight tremor of the tongue, and the pulse was 98 per minute. The question of alcohol was cautiously inquired into, but an absolute denial of excess was made. As, after waiting some time, the pulse did not become less frequent, the applicant was requested to call again the next morning. He did so, and, although his manner betrayed no nervousness, the pulse was again 97 or 98. He was advised to wait for a week and call again. He did so, with the same result as on the former occasions. His proposal was declined. Subsequent inquiries by the agent of the insurance company concerned established the fact that the applicant had been a heavy drinker.

Indulgence in morphia, cocaine, chloral, ether, antipyrin is sometimes concealed. The possibility of such indulgence should be borne in mind when dealing with obscure nervous conditions. The hard smoker usually confesses to his vice, which he regards as a venial one.

Finally, patients *place a false emphasis on certain symptoms*. Pain naturally bulks largely to the patient, while it is often a symptom of subordinate importance to the physician. In investigating pain, we should inquire into—

- (a) Its general character and intensity.
- (b) Its site and radiation.
- (c) Its recurrences or periodicity.
- (d) Its relation to food, exercise, sleep, emotion.
- (e) Its exciting causes, and means of relief.
- (f) Whether conjoined with tenderness or not.

Pain is often a symptom of small diagnostic value. We cannot weigh or measure it. The personal equation cannot always be solved.

There are some conditions, however, where a careful analysis of the pain present may go far to establish the diagnosis. Of such conditions I may enumerate the following: tabes dorsalis, cerebral tumour, sciatica, peripheral neuritis, gallstone colic, renal colic, gastric ulcer, aneurism, tumour of the spinal cord, trigeminal neuralgia.

When the patient complains of pain to the exclusion of other symptoms, pains in many organs, pains of variable but often great intensity, the suspicion of hysteria or neurasthenia is aroused.

While pain is usually fully dwelt on by the patient, he is often comparatively unobservant of far more significant symptoms, *e.g.* dyspnœa. Patients who are manifestly short of breath sometimes deny the fact, or regard it as of no consequence. Patients sometimes erroneously deny that they have any cough—a fact to be borne in mind when inquiring into the possibility of incipient phthisis. Night-sweating is also sometimes denied, or put down simply to the heat of the weather.

The condition of the bowels will often be inaccurately described, unless the practitioner is careful to obtain precise information. One of my former teachers was fond of a story—probably apocryphal, but not without

a useful moral—of a lady who consulted a doctor, and inquiry was made if the bowels were regular. “Quite regular,” was the reply. “May I ask how often they act?” said the doctor. “Oh, about once in three weeks”!

LECTURE III.

PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (A).

SUMMARY.

Posture of the Patient.

Methods of examination, viz. Inspection, Palpation, Percussion, Auscultation, Mensuration.

Inspection.

Its importance, mode of practising.

We inspect—

- (a) The condition of the integuments.
- (b) Alterations in the shape of the chest.
- (c) Alterations in the movements of the chest.

Palpation.

Mode of palpating, alterations in the vocal fremitus, Baccelli's sign, honchal fremitus, tussive fremitus, fluctuation, displacements of the cardiac impulse.

Percussion.

Mode of percussing, mediate and immediate percussion, light and deep percussion, staccato percussion, varieties of percussion-note, resonance and pitch; tympanitic, hyper-resonant, resonant, muffled, and dull notes; Wintrich's sign, Skodaic resonance, amphoric resonance, cracked-pot sound, sense of resistance, auscultatory percussion.

Percussion sounds in pleurisy, bronchitis, pulmonary collapse, emphysema, pneumonia, passive congestion and œdema, broncho-pneumonia, fibrosis, new growths, actinomycosis, hydatids, phthisis.

Posture of the Patient.

In chronic cases, where the patient's strength permits, he should be seated upon a common chair, with the chest fully exposed. He should be directed to let the arms hang by his sides, straighten the spine, slightly

elevate the chin, and open the mouth. He should then be instructed how to breathe—an act which few patients perform correctly without some preliminary drilling. Many patients breathe too shallow, others—especially males—breathe almost solely with the diaphragm, others, from nervousness, breathe in a jerky and unequal fashion. These errors can be rectified by a little patience on the part of the physician, who will find the time thus spent by no means wasted. In acute cases, and in presence of much prostration, it will be necessary to examine the patient in bed. The shoulders should be supported by an extra pillow, the head should also be supported, and the chest fully exposed as before. It cannot be too strongly insisted that no examination of the chest is adequate which does not embrace an examination of all its parts. We must on no account give an opinion in a pulmonary case until we have fully explored the chest in every part.

The methods of the physical examination of the lungs and pleura are as follows :—

- A. Inspection.
- B. Palpation.
- C. Percussion.
- D. Auscultation.
- E. Mensuration.

It will be convenient to take these methods in the above order. Succussion hardly deserves to be ranked as an independent method. It will be considered under auscultation.

A. Inspection.

The importance of inspection of the chest is, perhaps, hardly adequately recognized. It is a method of the highest importance, and should never be omitted.

The best point of view for inspecting the chest is a point on the level of the patient's thorax on the opposite side from that from which the light falls. The next best point of view is from behind, the observer looking over the patient's shoulders as he remains seated. The least satisfactory point of view is from the front. It is impossible to inspect the chest adequately from this point. It will greatly conduce to correctness of diagnosis to collect carefully all the information which can be obtained from inspection before proceeding to the other methods of examination.

By inspection we seek information regarding:—

- (a) The condition of the integuments.
- (b) Alterations in the shape of the chest.
- (c) Alterations in the movements of the chest.

(a) *The condition of the integuments.*—Among the conditions which may attract our attention are the following:—Œdema, venous turgescence, cyanosis, swellings, tumours, nodules.

(b) *Alterations in the shape of the chest.*—These alterations may be either physiological or pathological. The student should be prepared for rather wide departures from the physiological norm as regards the shape of the chest. Many irregularities in the shape of the bony framework of the chest are of no importance, and throw no light upon the state of the lungs.

Certain types of chest often associated with definite pathological conditions are well known, and are described in all the text-books. It is unnecessary to discuss them minutely in this place. Of these types I may mention the following:—

- The alar or winged chest—often tubercular.
- The pigeon-breasted chest—usually rickety.
- The barrel-shaped chest—usually emphysematous.

Curvature of the spine may modify the normal shape of the chest.

(c) *Alterations in the movements of the chest.*—These are of the highest importance. Most forms of pulmonary disease involve some interference with the normal expansion of the chest.

We may have to deal with the following conditions:—

Delayed expansion, flattening, immobility, bulging, recession.

Delayed expansion, especially of one apical region, is an important sign of early phthisis. It usually precedes definite flattening.

Flattening of the chest, as a whole, especially in its upper parts, is common in persons of feeble physique and imperfect pulmonary development, apart from actual disease.

Flattening of one apical region, the expansion on the opposite side being normal, is an almost certain sign of phthisis. It tells us definitely that there is shrinkage and imperfect expansion of one apex, and by far the commonest cause of this condition is tuberculosis. It may be masked by co-existing emphysema.

Immobility of the chest, as a whole, does not exist, such a condition being incompatible with life. Immobility of one entire side occurs in pneumothorax and large pleural effusion. Immobility of one or both bases may be due to pleural effusion, pneumonia, pulmonary collapse or to causes in the abdomen interfering with the descent of the diaphragm.

Pain may interfere with the free movements of the chest. This may be somewhat misleading in the case of pleurodynia.

Bulging may be due to pleural effusion (very rarely), or to aneurism or mediastinal growth.

Recession of the chest—as seen in the supraclavicular fossa, the suprasternal notch, the intercostal spaces, and the epigastrium—suggests some obstacle to the free entry of air into the lungs, with or without pulmonary collapse. Immobility of the diaphragm suggests paralysis of the diaphragm, diaphragmatic pleurisy, or such abdominal causes as peritonitis, ruptured gastric ulcer, etc.

B. Palpation.

Mode of palpating.—One hand should, as a rule, be used in palpating, and should be applied successively to different parts of the chest, while the patient pronounces the words “ninety-nine.” The two sides of the chest must in all cases be compared, as there is no fixed standard of conduction of the vocal fremitus, and we cannot draw any safe conclusions until we have satisfied ourselves of its normal condition in the case before us.

The vocal fremitus may be entirely absent in normal individuals, especially in women or children with thin, high-pitched voices, but this phenomenon is also found in vigorous and muscular subjects, in whom the thickness of the parietes seems to prevent the conduction of the vibrations to the hand.

The fremitus is more marked in health upon the right side than upon the left. The difference is usually quite distinct, and must be allowed for before any conclusions are drawn. There is an old clinical rule which says, “If the fremitus be equal on the two sides and well marked, suspect a lesion of the left apex. If the fremitus be equal on the two sides and ill marked, suspect a lesion on the right side.” In the former case we have probably to deal with an increase upon the left side, in the latter case with a decrease upon the right.

Vocal fremitus is diminished in thickened pleura, in

pleural effusion (serous, sanguineous, purulent), in hydrothorax, in certain cases of consolidation of the lungs (when the bronchi are collapsed or filled with secretion), in some new growths in the lungs, in pneumothorax.

Vocal fremitus is increased in most cases of consolidation of the lungs—pneumonic, tubercular, malignant. This increase seems to depend upon the bronchi remaining patent.

Baccelli's sign.—Baccelli holds that the vocal fremitus is better conveyed through serous than through purulent effusions, and that this difference possesses diagnostic importance. I believe this rule to be untrustworthy.

Friction fremitus is common in pleurisy, and may assist the diagnosis.

Rhonchal fremitus is common. It is not of much diagnostic importance.

Tussive fremitus is sometimes helpful in the diagnosis of pulmonary disease in children. It is to be interpreted on the same lines as vocal fremitus.

Fluctuation is a very rare sign. It is occasionally observed in connection with old empyemata.

The position of the cardiac impulse must in all cases be determined with the utmost care. Many grave errors are the consequence of neglecting this simple rule.

The principal deviations from the normal position of the impulse found in association with pulmonary disease are the following :—

1. Apex displaced to the left.

e.g. Right pleural effusion, old-standing left pleurisy involving cardiac adhesions, compensatory hypertrophy of the right lung depending on tubercular disease of the left, right pneumothorax, actinomycosis of right lung.

2. Apex displaced to the right.
e.g. Similar conditions on the two sides of the chest.
3. Apex displaced upwards.
e.g. Tubercular disease and retraction of left upper lobe.
4. Apex displaced downwards.
e.g. Tumour of left lung.

Let me emphatically endorse Kingston Fowler's dictum: "The position of the cardiac impulse is the key to the diagnosis of many affections of the chest."* The "strange cases of pneumonia" which turn out to be pleurisy or empyema, and are not recognized promptly because the position of the apex is not carefully ascertained, are only too familiar.

C. Percussion.

Our knowledge of percussion is due mainly to Auenbrugger, Corvisart, Lænnec, Piorry, Aran, Wintrich, Traube, and Gerhardt.

Percussion is the most difficult mode of physical examination of the chest. Prolonged practice is necessary to acquire even moderate skill in the practice of percussion. The method is, however, of the highest importance—hardly inferior in value to auscultation.

Pleximeters and hammers of all kinds are, in my judgment, useless, or worse. The fingers are adequate for all purposes. It is a matter of indifference whether the first or the second finger of the left hand be used as a pleximeter, and whether one, two, or three fingers of the right hand be used as a hammer. A very satisfactory note can be elicited by using the first finger of the left hand as pleximeter, and the second finger of the right hand as hammer. The stroke should be in every case from the

* Diseases of the Lungs, Fowler and Godlee, p. 54.

wrist—never from the elbow or the shoulder. It is well to vary the stroke, both as regards mode and intensity, *i.e.* more or less staccato, more or less forcible. The staccato note is very helpful in detecting the finer shades of dulness in the infraclavicular regions. A light stroke informs us as to the state of the parts immediately under the finger, a forcible stroke informs us of the condition of deeper or more distant parts.

The note elicited by percussion depends upon—

- (a) The condition of the chest wall as regards thickness and elasticity.
- (b) The amount and distribution of air in the parts under the percussing finger.

A thin chest wall, *cæteris paribus*, gives a more resonant note than a thick chest wall, an elastic than an inelastic wall.

The infraclavicular regions are commonly the most resonant part, the suprascapular regions the least resonant part, of the chest.

The supraclavicular regions can be most easily percussed from behind.

Skoda was probably right in holding that there is no such thing as a "liver note," "spleen note," "heart note," "stomach note," that all solid organs yield the same percussion note, and that in air-containing organs the note has relation simply to the amount of air which it contains. "The different sounds which percussion produces over the regions of the liver, spleen, heart, lungs, and stomach do not depend upon any peculiarities in these organs, but upon variations in the quantity, distribution, and tension of the air present in the regions in which they lie, and upon the force of the percussion stroke."*

* A Treatise on Auscultation and Percussion, J. Skoda, Markham's translation, pp. 6, 7.

We may recall that Piorry taught the contrary of this. I adhere to Skoda's doctrine.

Skoda * divides percussion sounds on the following plan—

1. Voll und leer (full and empty).
2. Hell und Dumpf (clear and dull).
3. Tympanitisch und nicht tympanitisch (tympanitic and non-tympanitic).
4. Hoch und tief (high-pitched and low-pitched).

He adds, "Ein voller Perkussionsschall kann hell oder gedämft, tympanitisch oder nicht tympanitisch, hoch oder tief, sein; eben so verhält es sich mit einem leeren Schalle."

The adequate criticism of this classification would take us far. Our point of view is not that of physics, but of practical medicine, and there seems no clear advantage in drawing distinctions which are hardly capable of application at the bedside.

Practically, a percussion note possesses—

- (a) Varying degrees of resonance.
- (b) Varying degrees of pitch.

"The sound (*i.e.* the sound produced by percussion) is a tone, clear or muffled, even to complete privation; this is the first and great distinction. And next, the tone is of a pitch higher or lower. Upon these two hang the whole theory and practice of percussion." †

Percussion sounds may be divided into—

- (a) Tympanitic, *e.g.* in pneumothorax.
- (b) Hyper-resonant, *e.g.* in emphysema.
- (c) Resonant, *e.g.* in healthy lung.
- (d) Muffled, *e.g.* in commencing consolidation.
- (e) Dull, *e.g.* in complete consolidation, or pleural effusion.

* Percussion und Auscultation, J. Skoda, p. 8.

† S. Gee, Auscultation and Percussion, third edition, p. 70.

The pitch of these notes progressively rises, beginning with the tympanitic note, of which the pitch is the lowest of the series.

The term "tympanitic" has been used in several different senses by various writers. The above application of the term is in accord with English usage, and is convenient. In certain cases—*e.g.* in pneumothorax, where the tension of the air in the chest gradually increases—a note previously resonant and low-pitched loses its resonance, and becomes almost dull. To such a note the term "tympanitic dulness" is sometimes applied.

As a rule, the more open the mouth the higher the pitch of the percussion note. This was first pointed out by Wintrich in connection with the percussion over cavities, and hence is sometimes known as "Wintrich's Sign."

Notes of varying degrees of dulness are found in the following conditions: Thickened pleura, pleural effusion, consolidation, infiltration, induration, compression, atelectasis.

A few special forms of percussion note remain for consideration, viz. Skodaic resonance, amphoric resonance, cracked-pot sound.

Skodaic resonance.—This is a note of resonant quality and of somewhat low pitch. This note is heard under the clavicle on the affected side in cases of pleural effusion. Various views have been held as to its mode of production. Skoda, by whom this note was first described, regarded it as due to relaxation of the lung. "That the lungs, partially deprived of air, should yield a tympanitic, and when the quantity of air in them is increased, a non-tympanitic sound, appears opposed to the laws of physics. The fact, however, is certain, and is corroborated both by experiments on the dead body, and also by this constant phenomenon, viz. that when the lower portion of the lung is

entirely compressed by any pleuritic effusion, and its upper portion reduced in volume, the percussion sound at the upper part of the chest is distinctly tympanitic." *

C. J. B. Williams thought this sound was produced by vibration of the air in the large bronchi, but this is an improbable view.

The higher pitch of Skodaic resonance, as compared with that present in pneumothorax, is probably due to the diminution of the vibrating area under such conditions.†

Amphoric resonance is a variety of tympanitic resonance sometimes heard in percussing over large and superficial cavities.

The cracked-pot sound (*bruit de pot fêlé*) is a well-known and valuable sign. It is a resonant note of variable pitch, metallic in quality, and characterized by a peculiar "chink," fairly well imitated by placing one hand over the other so as to form an air-containing space, and then smartly striking the knee with the under hand. The patient's mouth must be kept open. This sound is most often heard over cavities which are large, superficial, air-containing, and communicate freely with a bronchus. Most observers are agreed that a sound not easily distinguishable from the cracked-pot sound is sometimes heard under normal conditions, *e.g.* when percussing the back of a screaming baby. Skoda denied this. The cracked-pot sound may occasionally be heard over the resonant portion of lung in pleural effusion, and in cases characterized by incomplete consolidation of the lung, *e.g.* certain stages of pneumonia, tuberculosis, cancer. A typical cracked-pot sound is, however, strongly suggestive of cavity.

* A Treatise on Auscultation and Percussion, J. Skoda, Markham's translation, pp. 13, 14.

† Bristowe and others.

Sense of resistance felt in percussion.—The pleximeter finger conveys a sense of resistance which is to be distinguished from the sound produced in percussion. This sense of resistance was regarded by Piorry as of more value than the percussion sound—an untenable view—but its importance is hardly enough recognized. This sense of resistance attains its maximum in pleural effusion, provided the effusion is sufficient to make tense the pleural walls. Massive and dense pulmonary consolidations come next. Lung infiltrated with tubercle and commencing fibrinous exudation gives a less sense of resistance. In pneumothorax and emphysema the distension of the pleural cavity or of the air-cells may give rise to a certain degree of resistance. Healthy lung offers no sense of resistance. The amount of resistance depends mainly upon the degree of distension of the pleural cavity with fluid or with air, or upon the density of a pulmonary consolidation.

Auscultatory percussion.—This form of percussion has been advocated by some authorities as useful in mapping out the area of the heart and the liver.

I have never found it of practical service.

I shall now give a synoptical view of the various forms of percussion sound found in the different diseases of the lungs and pleura.

1. Pleural Effusion.

The degree of dulness depends not directly upon the amount of fluid in the pleural cavity, but indirectly upon the deprivation of the lung of air. An absolutely dull note shows that there is fluid present and that the underlying lung is airless.

In moderate effusions, where the lung still contains some air, the percussion note is muffled rather than dull.

The degree of dullness throws no light upon the character of the fluid. It is the same whether the fluid be passive effusion, serous exudation, pus, or blood. The dull area in pleural effusion often has a characteristic outline, viz. the upper limit is a curved line with its highest point in the axilla and the lowest point in front (Ellis's Curve).

In patients who have been confined to bed for some time the highest point of the dull area is usually posterior.

Posture, as a rule, has no influence upon the shape of the dull area. Exceptions to this rule sometimes occur, especially in passive effusions.

Dulness in pleurisy may be due to thickened pleura, and it is sometimes of considerable importance to distinguish this condition from pleural effusion. The distinction is not always easy. At the termination of an ordinary favourable case of acute pleurisy, when convalescence is complete, it is common to find slight dullness and some enfeeblement of the respiratory murmur at the affected base. This is due to thickened pleura, and is usually of no consequence. If this condition be mistaken for a residual effusion, errors both as regards prognosis and treatment will arise. How are we to distinguish a small residual effusion from a thickened pleura? Mainly by attention to the following points:—

- (a) The degree and extent of the dullness.
- (b) The condition of the vocal fremitus.
- (c) The position of the heart.

If the dullness be slight in degree and rather extensive in area, if the vocal fremitus be fairly well marked, and if the position of the cardiac impulse be normal, we are probably dealing with a thickened pleura. If the dullness

be marked, the vocal fremitus weak or absent, and (above all) if the heart be displaced, fluid is present.

The sense of resistance attains its maximum in large pleural effusions, and in solid growths of the lungs or pleura.

2. Pneumonia.

(a) *Stage of engorgement.*

The percussion note is either normal or more or less tympanitic. Skodaic resonance is occasionally present. There is no dulness.

(b) *Stage of consolidation.*

Dulness of varying degree is usually present.

The dull note may be modified by—

- (1) The presence of healthy lung tissue either above or beneath the consolidated area.
- (2) The presence of patches of air-containing lung intermingled with the consolidated areas.
- (3) The presence of air in a large bronchus beneath a thin layer of consolidated lung (Bäumler and V. Jürgensen).
- (4) The transmission of the stomach note through a consolidated left lower lobe anteriorly (Finkler).

The above causes account for the fact that a tympanitic percussion note (more rarely a normal resonant note) may be present during the consolidation stage of pneumonia. On the whole, however, dulness is fairly constant. All parts of the chest should be carefully explored. "Many pneumonias both of the upper and the lower lobe first show hepatization in the axillary region, and many pneumonias which begin centrally first reach the surface

in front or at the sides, and in this way become accessible to the methods of physical examination " (Gerhardt).*

The lung tissue in the neighbourhood of the consolidated area often yields a more or less tympanitic note.

(c) *Stage of resolution.*

Dulness usually becomes less marked, and gradually gives way to normal resonance. A clear, tracheal, tympanitic, or Skodaic note is sometimes heard during this stage.

3. Broncho-pneumonia.

The percussion sounds present in broncho-pneumonia are variable, and may be misleading. If the pneumonic foci be large and tend to become confluent, areas of dulness will be present. Dulness may also be due to collapse, which is frequently present.

On the other hand, over-distension of the air vesicles often gives rise to varying degrees of hyper-resonance. The percussion sounds may be normal.

Aufrecht is of opinion that dulness at the apices is sometimes due to lobular pneumonia confined to the bases. "These changes," he says, "in the percussion note over the normal apex can be explained only by the fact that, under the influence of the changes at the base, the tension of the normal tissue at the apices had been modified, so that the percussion note, as compared with the high tympanitic quality over the lower lobes, appeared empty and almost dull." †

4. Passive Congestion and Œdema.

These conditions may be fully present without any alteration of the percussion note.

* Nothnagel's Encyclop. of Practical Medicine, English edition, Art. Pneumonia, p. 459, quoted by Aufrecht.

† *Ibid.*, p. 568.

If the lung becomes practically airless, the percussion note will be dull, but it is necessary to insist that a high degree of congestion and œdema is consistent with normal percussion resonance.

In some cases of œdema we get the unduly clear note characteristic of relaxed lung.

Hydrothorax is often co-existent, and will give rise to dulness at the bases.

5. Pulmonary Induration (Fibrosis of the Lung).

Most of these cases are tubercular, but fibrosis may be due to irritative bronchitis, broncho-pneumonia, neoplasms, pressure by aneurism, foreign body in the bronchi, syphilis, and other causes.

Dulness may be present, but, on the other hand, a tympanitic or amphoric note may be heard, usually depending on bronchiectatic cavities.

6. Pulmonary Infarction.

If the infarct exceed a certain size, dulness (due directly to the infarct) will be present.

Dulness may also be due to a localized pneumonia set up by the infarct.

7. Bronchitis.

(a) Acute.

In acute uncomplicated bronchitis the percussion note is not necessarily affected. No amount of secretion in the bronchial tubes will cause dulness, so long as the lung tissue remains unaffected. Alterations of percussion resonance are, however, common in acute bronchitis, and depend upon some of the following conditions, viz.—

1. Pulmonary collapse.
2. Acute emphysema.
3. Relaxation of the lung tissue.

Collapse causes a muffled or dull percussion note. Acute emphysema and relaxation of the lung tissue give rise to varying degrees of hyper-resonance.

In the acute bronchitis of young children the practitioner should be watchful for the supervention of collapse.

(b) Chronic bronchitis and emphysema.

In these conditions the percussion note undergoes two changes, viz. it becomes more resonant and it falls in pitch. The area of pulmonary resonance is increased upwards and downwards. The superficial area of cardiac dulness is diminished or obliterated. Resonance may extend in front as low as the seventh rib, behind as low as the twelfth. In some cases there is dulness at the bases due to slight effusion. In very extreme cases of emphysema the percussion note may become less resonant and acquire a certain flatness of tone.

Biermer has described a percussion note in emphysema which he compares to the resonance of a paper-box, and which he calls "Schachtelton" (box-tone). This sound is produced by light percussion with the fingers, the pleximeter finger being placed in an intercostal space.

The percussion sounds in emphysema may be modified by rigidity of the chest, which in old subjects is sometimes well marked.

8. New Growths in the Lung.

Dulness is often present, and a hint as to diagnosis may sometimes be obtained by attention to the situation and limits of the dull area. This area is variable, but, unlike the dulness present in tuberculosis, is most often in the mid-region of the lungs. Its situation, mode of progress, and lines of extension present no analogy to the well-known rules which prevail in tuberculosis.

9. Actinomycosis.

Dulness is present, usually basic in situation.

10. Hydatids of the Lung.

The most typical percussion sound is dulness over a rounded area, absolute in the centre and becoming gradually less marked towards the periphery.

Deep-seated cyst may present a normal condition of things as regards percussion.

The portion of lung compressed by the cyst or cysts may yield the "relaxed lung" note.

11. Tuberculosis.

Attention to the percussion sounds is of the utmost importance in the diagnosis of pulmonary tuberculosis. Every variety of percussion sound may be present in this disease, but certain more or less definite rules can be formulated.

(a) *Acute miliary tuberculosis.*

The percussion sounds may be normal.

A slight degree of dulness, rarely marked, may be present.

Hyper-resonance, due to over-distention of the air vesicles, is common.

Alterations in the percussion sounds may occur from day to day.

(b) *Acute pneumonic phthisis.*

The percussion sounds resemble those of pneumonia.

(c) *Fibroid phthisis.*

See under Pulmonary Fibrosis.

(d) *Chronic fibro-caseous tuberculosis of the lungs*
(chronic pulmonary phthisis).

The percussion sounds may be normal. "The sound

of a lung containing merely a few solitary tubercles does not differ from that of a healthy lung" (Skoda).

Dulness, varying in degree, may be present, due to—

- (a) Abundant tubercular deposit.
- (b) Patches of pneumonia.
- (c) Fibroid thickening, especially round a cavity.
- (d) A fluid-containing cavity.

Hyper-resonance may be present, due to—

- (a) Relaxation of the lung tissue.
- (b) A cavity containing air and abutting on the surface.
- (c) Bronchiectatic dilatation of the tubes in relation with a tuberculous area.

In incipient phthisis careful percussion of the supra-clavicular, infraclavicular, and suprascapular regions is of the highest value. Slight degrees of dulness in these regions are very significant. The pitch of the note tends, I believe, to rise on the side where there is commencing infiltration.

Infiltration may proceed a considerable way before any dulness can be detected, and even extensive infiltration and softening may occur without any notable degree of dulness being present.

In some cases of infiltration and softening increased resonance is observed, due, probably, to relaxation of the lung tissue.

Percussion Sounds over a Pulmonary Cavity.

The following percussion sounds may be heard over cavities :—

- (a) Normal resonance, when the cavity is small or covered by a considerable layer of healthy lung.
- (b) Dulness, when the cavity contains fluid or is surrounded by a fibrous envelope.

- (c) Cracked-pot sound, when the cavity is fairly large, is near the surface, contains air, and communicates freely with a bronchus.
- (d) Hyper-resonance, when the cavity is large, superficial, and full of air.

The percussion note over a bronchiectatic cavity is usually resonant, owing to co-existing emphysema.

The most characteristic percussion sounds over a cavity are dulness and the cracked-pot sound.

The pitch of the percussion note is higher when the patient's mouth is kept open (Wintrich).

The percussion sounds give no information as to the pathological cause of cavity. They are conditioned solely by physical laws.

LECTURE IV.

PHYSICAL EXAMINATION OF THE LUNGS AND PLEURA (B).

SUMMARY.

History of auscultation, Lænnec's *Traité de l'Auscultation Médiate*.
Skoda's criticisms.

Methods of auscultation—mediate and immediate auscultation,
stethoscopes.

Accessory and extraneous sounds, their characters and diagnosis.

Auscultation of—

A. The breath sounds.

B. Adventitious sounds.

C. The voice.

A. The Breath Sounds.

Theories regarding the production of the vesicular murmur,
variations of the vesicular murmur in health and in disease.

Alterations as regards (a) Quality, (b) Intensity, (c) Rhythm ;
alterations in quality—Lænnec's and Skoda's classifications ;
vesicular breathing, bronchial breathing, cavernous breath-
ing, harsh breathing, weak breathing, delayed inspiration,
prolonged expiration, cog-wheel breathing.

B. Adventitious Sounds.

Pleural friction, rhonchus ; râles—classification of, bronchial
râles and cavernous râles, bubbling râles and crepitant
râles, size of râles, conduction of râles, metallic tinkling,
amphoric echo, succussion splash, *bruit d'airain*, post-
tussive suction.

C. The Voice.

Bronchophony, pectoriloquy, ægophony.

Mensuration.

AUSCULTATION, as a systematized art, is the creation of
Lænnec. Hippocrates had discovered the succussion
splash, and probably also pleural friction, but no substantial

progress was made from his day until that of the great French clinician. Lænnec, having discovered the principle of mediate auscultation, proceeded to work the subject to the bottom at the Hôpital Necker, Paris, and practically exhausted it, so far as the lungs are concerned. He was less successful with the auscultation of the heart. The most important criticism of Lænnec's doctrine was that of Skoda, who brought the laws of physics to bear upon the phenomena, and corrected and regularized Lænnec's teaching. Skoda's work was, in the main, critical rather than creative.

Lænnec's *Traité de l'Auscultation Médiate* will always remain one of the great landmarks of medicine, and should be read by every physician who interests himself specially in the diseases of the lungs and the heart. It is illuminated by profound insight and philosophic acumen, and is the fruit of vast experience and unwearied labour. It can never become antiquated, and subsequent observers are reduced simply to the position of critics. The great fundamental creative work has been done, once for all, by Lænnec. Nevertheless, the work is open to criticism from at least two points of view. Lænnec fell into two errors. He over-rated the importance of mediate auscultation, and he formulated a terminology of auscultatory terms too elaborate for application at the bedside, except for the specialist. If auscultation is to be made fully available for the practitioner who is not a pulmonary expert, it must be simplified. The present-day terminology of auscultatory pulmonary signs is in many respects diffuse and vague, over-elaborate and yet wanting in precision, confusing to the learner, and not readily handled by the specialist.

METHODS OF AUSCULTATION.

Auscultation is either (*a*) Immediate or (*b*) Mediate. In the former case the ear is applied directly to the chest-wall; in the latter some form of stethoscope is employed. Immediate auscultation, contrary to the opinion of Lænnec, is a method of great value. For obvious reasons, it is not always available. It is more suitable in the case of children than in that of adults, and more applicable to the posterior than to the anterior surface of the chest. A very satisfactory examination of the chest can often be made by the direct application of the ear, a thin handkerchief or towel being allowed to intervene between the ear and the thoracic wall. Guttman advises immediate auscultation in the case of feeble patients, and in exhausting diseases where it is important to go over the chest rapidly. Immediate auscultation is inferior to mediate in cases where precision is required, and where the definite localization of pulmonary signs may be of importance.

Mediate auscultation is the usual method. It is generally more convenient than immediate auscultation, more agreeable to the patient, more precise as regards topographical details, and more readily lends itself to the exploration of difficult and obscure signs. Every form of stethoscope, however, modifies the signs in some—but not usually in any important—degree. As to the form of stethoscope, the student will do well to familiarize himself with the wooden stethoscope, the binaural and the phonendoscope. For ordinary purposes the binaural stethoscope is the most convenient. It is immaterial which of the many varieties of this stethoscope we employ—perhaps the simpler the better. Care should be taken in adjusting the binaural to see that the ear-pieces fit easily into the ears and do not exercise any undue pressure. The

phonendoscope, which I have found of real utility in the exploration of obscure cardiac murmurs, is seldom of assistance in pulmonary cases. The chest-piece of the stethoscope should be applied lightly and evenly to the chest without pressure. The patient is then instructed to open the mouth, to inspire and expire fully and deeply without violent effort, and not to make any noises with the mouth.

Accessory and extraneous sounds.—Our first care in auscultation is to exclude or ignore certain sounds which do not arise in connection with the lungs and pleura. Sounds may be generated by imperfect coaptation of the chest-piece to the chest-wall, by inequalities of the surface of the chest, by hairs, by contact with clothing, by contraction of the muscles. These sources of error can usually be obviated without difficulty. The most serious difficulty of this kind arises in connection with abundant hair. A little oil applied to the surface mitigates this difficulty. Extraneous sounds can often be readily appreciated by the practised ear. They are often “superficial,” rubbing or scraping in quality, and do not conform to any recognized variety of pulmonary sound. It must be admitted, however, that the distinction is not always easy. A comparison of the sounds produced over different parts of the chest often gives us the clue to extraneous sounds.

Auscultation must be conducted upon a definite method. The most convenient method is the following. We listen in order to—

A. The breath sounds.

B. Adventitious sounds.

C. The conduction of the voice (vocal resonance).

It is most important to attend to these separately and in this order.

A. The Breath Sounds.

Normal vesicular breathing has a peculiar and well-known rustling or sighing quality. It varies much in intensity, according to the thickness of the parietes and the vigour of the respiratory act, inspiration (*i.e.* the audible inspiration) is four or five times as long as expiration, expiration is sometimes weak and may even be inaudible. In normal breathing the inspiration overshadows the expiration. The contrary is sometimes the case in certain states of disease.

The precise mode of production of the vesicular murmur is not certain. Three theories have been propounded on this subject, viz.—

- (1) That the vesicular murmur is produced in the alveoli by friction of the air against the walls of the alveoli at the moment of their dilatation (Lænnec, Skoda).
- (2) That the vesicular murmur is produced at the glottis, and is modified by conduction to the alveoli (Beau, Baas, Gee).
- (3) That the vesicular murmur is due to vibration of the lung tissue, its tension being increased during inspiration (Gerhardt).

It is important for the learner to make himself thoroughly conversant with the variations in the vesicular murmur which are consistent with a normal condition of the lungs. These variations are many.

The vesicular murmur varies in normal conditions—

- (1) According to the thickness of the parietes. *Cæteris paribus*, it is louder in thin than in stout or muscular subjects, louder where the chest coverings are thin—*e.g.* the infraclavicular regions—than where they are thick—*e.g.* the supraspinous regions.

(2) According to the thickness of the subjacent layer of lung ; louder, therefore, over the front and back generally than at the apices or the anterior or inferior borders.

(3) According to the activity of the respiratory process.

The intensity of the vesicular murmur sometimes varies on the two sides of the chest. These variations may have their basis in anatomical peculiarities, *e.g.* in variations in the shape, thickness, or direction of the bronchial tubes.

In the right infraclavicular region the vesicular murmur is usually more distinct than in the left, may be harsh or even bronchial in quality, especially in children. These physiological differences must be allowed for in investigating the signs of disease.

The breathing of the child is more *shrill* than that of the adult. It is called *puerile* breathing. The explanation is to be found chiefly in the thinness of the chest-wall in the child—in a minor degree, perhaps, in the greater elasticity of the lungs, and the greater activity of the respiratory process in childhood.

When we hear vesicular breathing of normal quality, we conclude, not that the lungs are necessarily healthy, but that air is entering freely. Normal breathing is not inconsistent with disease, *e.g.* a slight degree of bronchitis, a few scattered tubercles, the early stage of pneumonia. Upon the whole, however, when we have satisfied ourselves that the breathing is perfectly normal, we have gone a long way towards assuring ourselves that the lungs are healthy.

In conditions of disease the breathing may be altered as regards—

(a) Quality.

(b) Intensity.

(c) Rhythm.

(a) *Alterations in quality.*

The breathing may be vesicular, broncho-vesicular, bronchial, tubular, amphoric, interrupted (cog-wheel, wavy).

It is desirable to simplify this classification, if possible. Omitting the case of interrupted (cog-wheel) breathing for the moment, inasmuch as it is a special case, and not liable to confusion with the other varieties, we may with substantial accuracy and great convenience regard the types of breathing as three in number, viz.—

1. Vesicular.
2. Bronchial (tubular).
3. Cavernous (amphoric).

Lænnec's classification of the varieties of breathing was as follows :— *

1. Respiration vésiculaire (normal vesicular breathing).
2. Respiration bronchique (bronchial breathing).
3. Respiration puérile (puerile breathing).
4. Respiration caverneuse (cavernous breathing).
5. Respiration soufflante (a variety of bronchial or, cavernous breathing, in which the air seems to be drawn towards the ear of the observer during inspiration and repelled from it during expiration).
6. Souffle voilé (veiled puff, described as a condition in which the air seems to set in motion a veil interposed between the ear of the observer and a cavity).

Skoda † subjected the above classification to a trenchant criticism, and had no great difficulty in showing that it was unsatisfactory.

He elaborated his own scheme as follows :—

1. Pulmonary respiratory murmur.

* Auscultation Médiante, Troisième édition, p. 46 *et seq.*

† A Treatise on Auscultation and Percussion, Markham's translation, p. 90 *et seq.*

2. Bronchial breathing.
3. Amphoric echo and metallic tinkling.
4. Indeterminate (*unbestimmte*) respiratory murmur.

Lænnec's classification seems open to criticism from several points of view. The *respiration soufflante* and the *souffle voilé* are simply varieties of cavernous breathing, not sufficiently distinctive to merit separate enumeration. The *respiration soufflante*, as described by Lænnec, is a familiar phenomenon. On the other hand, the *souffle voilé* has not been generally recognized.

Skoda's classification seems also somewhat faulty. His "indeterminate breathing," by which he understood a variety of breathing neither purely vesicular nor definitely bronchial, is a mere negative term, incapable of strict scientific application. "Amphoric echo" and "metallic tinkling," are not genuine types of breathing. These phenomena, which are not of the first importance, are better considered as adventitious sounds.

Again, Skoda denied that there was any real distinction between bronchial breathing and cavernous breathing. In this view he seems to have been mistaken. Cavernous breathing has a definite quality of its own, which can be recognized, and from which important information may be obtained.

The breathing, then, in states of disease may be—

- i. Vesicular.
- ii. Bronchial.
- iii. Cavernous.

i. *Vesicular breathing.*

Vesicular breathing, normal in all respects, may, as already stated, be present in disease. Oftener it is altered as regards (*b*) Intensity or (*c*) Rhythm.

(b) *Alterations in intensity.*

Exaggerated or harsh vesicular breathing may occur in—

1. The early stage of bronchitis, where it is due to slight swelling of the bronchial mucous membrane.
2. Commencing phthisis, where it is due to localized catarrh, usually of one apex.
3. In the early stage of pneumonia, before consolidation has begun (Stokes).
4. Over the sound side or sound portion of an affected side, when from any cause (pleural effusion, pneumonia, tuberculosis, infarction) one side, or a portion of one side, is deprived of its functional activity.
5. In certain conditions of dyspnœa, depending upon disease in organs other than the lungs, *e.g.* heart, larynx.

Diminished or weak vesicular breathing may occur in—

1. Pleural effusion.
2. Bronchitis and emphysema.
3. Pneumonia before consolidation is established and during resolution.
4. Phthisis.
5. Cancer.

The causes of weak breathing in the above cases are sufficiently obvious. They are as follows:—

- (a) Pressure upon the lung from without, *e.g.* by pleural effusion.
- (b) Impediment to the free entry of air into the lungs.
- (c) Diminished elasticity of the lungs.
- (d) Pressure upon the lung by solid growth in the lung.

Several of the above causes may co-exist.

Harsh breathing should suggest to our minds either—

- (a) That there is a slight obstruction to the entrance of air into the vesicles of the part; or
- (b) That a portion of the lung is abnormally active, owing to deficiency of activity in other portions of the lungs.

Weak breathing should suggest to our minds either—

- (a) That there is a considerable obstruction to the entrance of air into that portion of the lungs; or
- (b) That the elasticity of the lung is diminished; or
- (c) That the lung is in part infiltrated or compressed.

Vesicular breathing may be altered as regards rhythm.

(c) *Alterations in rhythm.*

1. The inspiration may be “delayed,” *i.e.* the inspiratory sound may follow the inspiratory movements after a distinct interval.

The causes of this phenomenon are not well understood.

2. The expiration may be prolonged.

Prolonged expiration is characteristic of emphysema and of asthma. It is also heard over a limited area in commencing phthisis.

3. Inspiration or expiration or both (most usually inspiration) may possess the cog-wheel quality.

The significance of cog-wheel respiration has been much debated. When this sign occurs generally over the chest it may be ignored. When it is limited to one or both apices the suspicion of commencing phthisis may arise, but in the absence of other signs no definite conclusion can be drawn. I have sometimes found this sign very fluctuating, appearing and disappearing in the course of the same examination. Its causes are many—nervous, muscular, pulmonary. It cannot be denied that it is a

common sign in commencing phthisis, but it is not to be relied upon in any case of doubt.

ii. *Bronchial breathing.*

Bronchial breathing may be defined as a variety of breathing present in most healthy persons (especially in thin people) at the level of the seventh cervical and upper five or six dorsal vertebræ.

An ill-defined bronchial breathing may be heard about the root of the lung in the interscapular region, and in the right infraclavicular region in some persons. To this form of respiration the term broncho-vesicular may be applied.

Tubular breathing, strictly defined, does not occur in health. It is characteristic of somewhat dense consolidations, and resembles bronchial breathing with a superadded metallic quality.

Bronchial breathing has a peculiar quality to which the terms hollow and reverberating have been applied. It may be loud or weak, high-pitched or low-pitched. In the large majority of cases the special "bronchial" quality is more evident in expiration than in inspiration. The expiration is often of higher pitch than the inspiration. Whereas in normal breathing the expiration is short, weak, of a "dying-away" quality, and of lower pitch than the inspiration, in bronchial breathing these characteristics are reversed. Here the expiration is the pronounced element. It is usually well-marked, prolonged, often loud, of higher pitch than the inspiration, and of a peculiar hollow quality, readily appreciated when the ear has become familiarized with the sound. In studying bronchial breathing, the beginner in auscultation should fix his attention in a special manner upon the expiration.

Two theories are held regarding the mode of production of bronchial breathing :—

- (a) The doctrine of Lænnec, viz. that it is produced in the bronchi, and conveyed to the ear by the superior conducting power of solidified pulmonary tissue ; and,
- (b) The doctrine of Skoda, viz. that it is due to *consonance*, the bronchial respiration being generated or magnified in cavities and in the bronchi of condensed lung tissue by the air in these cavities and bronchi vibrating in consonance with that in the trachea.

Bronchial breathing occurs outside the physiological areas in the following conditions :—

1. In consolidation of the lungs (whether due to fibrinous exudation, tubercle, induration, fibrosis, neoplasms, or infarction), provided the main bronchi remain open.
2. In collapse of the lungs.
3. In many cases of pleural effusion, especially in children.
4. In certain cavities, whether tubercular or bronchiectatic.

We shall consider these conditions in order.

1. *Consolidation of the lung*.—Bronchial breathing gives us no hint as to the pathological nature of the consolidation. It does suggest to us a clue as to the density of the consolidation. The more dense the consolidation, the more pronounced the bronchial breathing. In very dense consolidations (*e.g.* in many cases of pneumonia) the bronchial breathing assumes the tubular type. In loose consolidations, or where dense consolidations are breaking up, the bronchial breathing present tends to become vaguely blowing and weak. In cases of consolidation, as already mentioned, patency of the main bronchial tubes is necessary for the production of bronchial breathing. If

the bronchial tubes become filled with secretion, bronchial breathing ceases. This is not infrequent in pneumonia, and may give rise to the unfounded conclusion that a sudden pleural effusion has set in. Attention to the position of the cardiac impulse will help to obviate this error.

2. *Pulmonary collapse*.—The bronchial breathing present in this condition is usually weak and "distant" in quality. Diagnosis is assisted by the co-existing retraction of the side, and by the diminution of the vocal fremitus and vocal resonance.

3. *Pleural effusion*.—In the majority of cases of pleural effusion bronchial breathing is audible at some stage of the process in the interscapular region. The explanation is the retraction of the lung towards its root, in consequence of the effusion. These cases are well known, and need give rise to no difficulty. Much more important are those cases (by no means rare) where bronchial breathing is heard over the entire affected side in pleural effusion. These cases are commoner in children than in adults. The explanation in bronchial breathing in this connection is not difficult. Fluid is a good conductor of sound, and if the large bronchi remain patent there is no reason why bronchial breathing should not be present in pleural effusion. The question arises, Can the bronchial breathing of pleural effusion be distinguished from the bronchial breathing of pneumonic consolidation? Skoda answers this question in the negative. Probably this answer is in strictness correct, but I have an impression that the bronchial breathing of pleural effusion rarely has the loud, insistent quality which is common in pneumonic consolidation, that it usually seems more removed from the ear, and that the expiration has its peculiar characters less marked than in cases of consolidation. In such cases we must pay

special attention to the state of the fremitus and the position of the heart.

Bronchial breathing is heard over cavities of moderate size, abutting on the surface, containing air, and communicating with a bronchus. It is sometimes best heard after coughing. The distinction between bronchial breathing and cavernous breathing, though often obvious, is sometimes very difficult, and the most experienced ear may sometimes be in doubt upon this point.

The intensity and the rhythm of bronchial breathing present many variations, but definite rules upon these points can hardly be formulated.

iii. *Cavernous breathing.*

Cavernous breathing resembles bronchial breathing, but has, in addition, a peculiar echoing quality due to consonance in a cavity.

To give rise to cavernous breathing, a cavity must be of considerable size, contain much air, be somewhat superficial in position, and communicate freely with a bronchus.

Some observers are of opinion that in cavernous breathing the expiration is always of lower pitch than the inspiration, while in bronchial breathing the reverse is the rule. After much attention to this subject, I have not been able to satisfy myself as to the validity of this rule.

Amphoric breathing may be considered as a variety of bronchial breathing. It has a peculiar metallic quality, comparable to the sound produced by blowing into a large empty bottle. It is characteristic of large cavities.

Cavernous breathing is occasionally present in cases of pleural effusion, especially in the mammary region.

B. Adventitious Sounds.

The subject of adventitious sounds in the chest is one of the most difficult in the whole range of medicine.

The terminology of the subject is unsettled and confused, and the learner is bewildered by a host of terms, incapable of precise definition or of easy application at the bedside.

I shall approach the subject from the point of view of the mechanism or mode of production of these sounds.

Two sounds are readily differentiated, and need not give rise to much difficulty, viz. pleural friction and rhonchus (*"le râle sec sonore ou ronflement"* of Lænnec, *"das trockene sonore und das trockene pfeifende Rasseln"* of Skoda).

Pleural friction is usually a rubbing or scraping (rarely crepitant) quality ; it is generally heard both during inspiration and expiration ; it is sometimes increased by pressure of the stethoscope ; it is usually (but not always) accompanied by the subjective sense of pain ; it may occur in any part of the chest, but is commonest in the inferior lateral and antero-lateral regions ; it is due to the rubbing together of the inflamed pleural surfaces.

Rhonchi are snoring or sibilant sounds, usually heard both during inspiration and expiration, possessing a definite musical quality, varying in pitch according to the calibre of the tube in which they are produced, sometimes disappearing or changing their position after coughing.

Rhonchi are produced by the narrowing of the normal calibre of a bronchiole, either from the presence of mucus or of inflammatory swelling of the mucous membrane.

Rhonchi are (a) Sonorous, and (b) Sibilant, the former produced in the large or middle-sized, the latter in the small, tubes.

Pleural friction and rhonchi are usually recognized without much difficulty.

It is otherwise with the large group of sounds to which such terms as the following are applied: true crepitus, crepitant râle, sub-crepitant râle, muco-crepitant râle, mucous râle, bubbling râle, consonating râle, gurgling râle, indeterminate râle, and so forth.

Can we see any light amidst this bewildering catalogue of ill-defined terms?

Let us inquire what is the *sine quâ non* for the production of an adventitious pulmonary sound, which is neither a friction sound nor a rhonchus.

It is probably to be found in two conditions, viz.—

- (a) The presence of fluid in a bronchiole or a cavity; and
- (b) The passage of air through this fluid.

It has often been assumed that râles of a certain type are produced in the air vesicles, and the so-called “true crepitus” of the first stage of pneumonia, and the fine râle of œdema and of pulmonary collapse, are supposed to be cases in point. Much attention has been bestowed upon the fine crepitus of commencing pneumonia, and the usual doctrine is that this sound is produced in the air vesicles by the entry of air into them at the time when fibrinous exudation is just beginning. Does this doctrine rest upon any real foundation? I think not. The more we reflect upon the subject the more doubtful does it become whether the entry of air into a vesicle filled or filling with fibrinous exudation affords the conditions necessary for the production of this sound. Hence, an increasing number of observers* are inclined to regard the so-called “true crepitus” of the first stage of pneumonia as really due to pleural friction.

I adopt this view, and suggest the following considerations in its support:—

* See Osler, *Practice of Medicine*, fourth edition, p. 119.

- (a) The sound has sometimes a distinctly rubbing quality.

This statement, no doubt, begs the question at issue and must be taken simply as an expression of opinion.

- (b) It is "superficial" rather than deep.
- (c) It bears no close resemblance to the fine crepitations of capillary bronchitis, or miliary tuberculosis.
- (d) It is usually accompanied by pain.
- (e) It is often absent—a fact more easily understood on the pleural than upon the pulmonary view of its causation.

That a fine crepitating râle is produced by the opening up of collapsed air vesicles is probable, but in such cases it is quite conceivable that the sound is really produced in the terminal bronchiole, rather than in the air vesicle.

If the above reasoning is sound, we have to deal with two kinds of râle, viz. those produced in a bronchiole and those produced in a cavity. About the mechanism of the latter râle there is no difficulty.

Râles, then, are (a) Bronchial, and (b) Cavernous.

The quality of bronchial râles depends on the following considerations, viz.—

- (a) The size of the tube in which the râle is produced.
- (b) The amount and character of the fluid in the tube.
- (c) The condition of the surrounding lung tissue.

The quality of cavernous râles depends on the following considerations, viz.—

- (a) The size of the cavity,
- (b) The condition of its walls.
- (c) The amount and character of the fluid in the cavity.
- (d) The degree of communication between the cavity and the bronchi.

Bronchial râles.

(a) According to the size of the tube in which the râle is produced, the râle will be large, medium, or small (fine).

(b) According to the amount of fluid in the tube, the râle will be moist or dry. These terms are unfortunate, but they have the sanction of long usage. In this connection "dry" is only a relative term. It means "less moist."

(c) According to the condition of the surrounding lung tissue, the râle will be either—

1. Bubbling, when the surrounding lung tissue is spongy and air-containing ; or,
2. Crepitant, when the surrounding lung tissue is consolidated or fibrosed.

We have thus the basis of a rational classification if our assumptions are granted.

Bronchial râles may be divided into—

- (a) Large, medium-sized, or small (fine).
- (b) Moist or dry.
- (c) Bubbling or crepitant.

For example—

- (1) We hear a large, moist, bubbling râle.

Inference—that in a large bronchiole under our ear fluid is present, that the fluid is abundant, that air is entering the bronchiole, and that the surrounding lung tissue is spongy and air-containing.

- (2) We hear a fine, dry, crepitant râle.

Inference—that in a small bronchiole under our ear fluid is present, small in amount, that air is entering the bronchiole, and that the surrounding lung tissue is more or less consolidated.

That the quality, as well as the amount, of the fluid has

some effect upon the characters of a râle is highly probable, but we have no definite knowledge on the subject. Certain so-called "sticky," "creaking," and "rumbling" râles may be due to some special viscosity of the fluid.

Cavernous râles.

According to the size of the cavity, râles produced in it will be either less hollow or more hollow.

According as the cavity is surrounded by a fibrous envelope, or by spongy lung tissue, the râles will be bubbling or crepitant.

According as the amount of fluid in the cavity is large or small, the râles will be moist or dry.

In a large proportion of cases the cavity is fairly large, possesses a fibrous envelope, and contains a considerable amount of fluid; hence the râles are often large, hollow, crepitant, and moist. To this combination of characters the term "gurgling" is usually applied.

But the râles produced in a cavity may be crepitant, or bubbling, or "croaking" in quality.

The question arises whether the point at which a rhonchus or a râle is heard is any true index of the point at which it is produced. Lænnec's opinion on this point was as follows:—

"Certains râles, quoique très-forts, peuvent n'être pas entendus à un ou deux pouces du point où ils ont leur siège. Cela a surtout lieu pour le râle muqueux et le râle crépitant. Le ronflement, au contraire, et le râle sibilant s'entendent quelquefois d'un côté à l'autre de la poitrine, et par cette raison, ils compliquent souvent les autres espèces." *

In a doubtful case, it is well to weigh the possibility that a certain adventitious sound may not be produced under the ear, but at some more distant point.

* L'Auscultation Médiate, vol. i. pp. 109, 110.

There remain for consideration a group of adventitious sounds of minor importance, viz.—

- (a) Metallic tinkling.
- (b) Amphoric hum.
- (c) Succussion splash.
- (d) Bruit d'airain, or coin sound.
- (e) Post-tussive suction.

These sounds have this point in common—that they are heard only in connection with air-containing cavities, pleural or pulmonary.

(a) *Metallic tinkling (tintement métallique).*

This sound was compared by Lænnec* to the sound produced when a metal or porcelain cup is lightly struck by a pin, or when a grain of sand is dropped into it. This sound may be heard when the patient breathes, speaks, or coughs. Lænnec regarded this sound as indicative either of a serous or purulent pleural effusion combined with pneumothorax, or of a large pulmonary cavity partially filled with very liquid pus.

(b) *Amphoric hum (bourdonnement amphorique).*

This sound is regarded by Lænnec as a variety of metallic tinkling. He compares it to the sound produced by blowing into a carafe or pitcher. It may be heard in connection with the cough, the breathing, or the voice. This sound is indicative of a large air-containing cavity.

(c) *Succussion splash.*

This sound was known to Hippocrates. It is produced by seizing the patient by the shoulders and giving him a sudden shake, when a splashing sound is heard by the auscultator.

For the production of this sound the presence of air and fluid in the pleural cavity is requisite.

* L'Auscultation Médiate, vol. i. p. 113.

Succussion splash is usually easy of recognition. In exceptional cases splashing sounds in the stomach may give rise to difficulty.

(d) *Bruit d'airain, or coin sound.*

This well-known sound is produced by placing a penny or half a crown in contact with the chest and striking it with another similar coin. A peculiar chiming sound is produced in certain cases.

This sound is heard most typically in pneumothorax, but may also occur in connection with large pulmonary cavities.

(e) *Post-tussive suction.*

This is a peculiar sucking sound sometimes heard over cavities during inspiration. It is somewhat rare.

C. Vocal Resonance.

When we listen through the stethoscope while the patient speaks, the voice conduction may be modified in the following ways:—

- (a) It may be increased or diminished in loudness.
- (b) It may be increased or diminished in distinctness.
- (c) It may be altered in tone or quality.

It is impossible in the present state of our knowledge to keep these elements entirely distinct.

By bronchophony we understand that condition in which the voice is conveyed with increased intensity and heightened pitch, and with either increased or diminished distinctness.

By pectoriloquy, following Lænnec and the French school, we understand that condition in which the voice is conveyed, as it were, directly into the tube of the stethoscope. The voice is not louder than in health—may be much less loud—but it has always the quality of abnormal distinctness and direct conduction to the ear.

By ægophony we understand a peculiar bleating or twanging quality of voice, occasionally heard in the inter-scapular region in cases of pleural effusion.

Lænnec distinguished bronchophony from pectoriloquy, and taught that this latter sign was pathognomonic of cavity. Skoda denied that there was any real distinction between bronchophony and pectoriloquy. He also denied that pectoriloquy was pathognomonic of cavity. In this contention he was, no doubt, correct.

Bronchophony.

The causes of bronchophony are the same as those of bronchial breathing, and the two signs are always associated. Bronchophony occurs under the following conditions :—

1. In all conditions in which the lung parenchyma is deprived of air and rendered more or less solid, and in which the bronchial tubes remain patent, *e.g.* pneumonia, tuberculosis, pulmonary fibrosis, pulmonary infarction, cancer.

“Pneumonia in its first stage, inflammation confined to a few lobules of the lung, lobular hepatization, œdema of the lungs, or limited effusion of blood into the lung parenchyma produce very slight or no increase whatever in the strength of the thoracic voice ; neither do solitary tubercles, however numerous, provided the intervening parenchyma remains pervious to air” (Skoda).*

2. In cavities of a certain size, which contain air and communicate freely with a bronchus.
3. In certain cases of pleural effusion.
4. In pulmonary collapse.

Pectoriloquy, as Lænnec taught, is common over

* *Op. cit.* p. 44.

cavities, but it is not invariable in such conditions, and it is heard in some cases of consolidation. Pectoriloquy suggests the probability of cavity, but cannot be relied upon apart from other signs. Flint thought that when bronchophony and pectoriloquy existed together consolidation was present, but that from pectoriloquy alone a cavity might be inferred.

Ægophony probably indicates a thin layer of pleural effusion. It is a rare sign, seldom found in its typical form, of doubtful mechanism, and of little practical value in diagnosis.

Speaking generally, increased vocal resonance suggests to us the probability of either—

- (a) Pulmonary consolidation (pneumonic, tubercular, cancerous, etc.), the bronchial tubes remaining patent; or
- (b) A cavity.

Diminished vocal resonance suggests to us the probability of either—

- (a) Fluid in the pleural cavity.
- (b) Thickening of the pleura.
- (c) A very dense form of pulmonary consolidation.
- (d) Blocking up of the bronchial tubes with secretion.
- (e) Pressure on the lungs (including the tubes) by tumour or aneurism.

Mensuration.

Mensuration is important, as giving us a gauge of pulmonary capacity and expansion. Accurate measurements are not easily made, nor has the cyrtometer proved of much practical utility. The semi-circumference of the right side of the chest usually exceeds that of the left by from half an inch to an inch. The semi-circumference of the affected side in pleural effusion may exceed that of the

sound side, but this rule is by no means universal, and cannot be relied upon.

The following passage from Gee's well-known work sets forth clearly the difficulties of drawing conclusions from chest measurements: "Circumferential measurements of the two sides are often made, but be it remembered, first, that considerable increase in the sectional area of the chest may occur, and the length of the periphery remain the same, by the passage of the elliptical form into the circular; and, next, that the displacement of the mediastinum, which accompanies unilateral enlargement, thrusts the heart into the unaffected side. And this consideration, too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side; and then it will be plain why, as a matter of fact, the perimeter of the expanded side often measures very little more, nay, even less, than that of the side which is not diseased." *

The periodic measurement of the chest at the mammary line is to be recommended as a good routine practice in pulmonary phthisis. It gives valuable information regarding the progress of the case.

Increased expansibility suggests the following possibilities in phthisis:—

- (a) Compensatory hypertrophy of the sound lung.
- (b) Emphysema in the neighbourhood of the diseased areas.
- (c) Pneumothorax.
- (d) Pleural effusion.

Diminished expansibility suggests the following possibilities:—

- (a) Progress of the disease.
- (b) A tendency to fibrosis and chronicity of course.

* Auscultation and Percussion, S. Gee, third edition, p. 29.

In pneumothorax the semi-circumference is usually increased on the affected side.

In thoracic tumour the semi-circumference is usually increased on the affected side.

I append a brief syllabus of the auscultatory signs found in the principal affections of the lungs and pleura.

1. *Acute bronchitis.*

(a) Breath sounds.

Quality—vesicular.

Intensity—normal, harsh, weak, abolished (collapse).

Rhythm—normal, expiration prolonged.

(b) Adventitious sounds.

Sonorous or sibilant rhonchi.

Bubbling râles (large, medium, fine).

Crepitant râles, only if complications involving condensation of lung tissue are present.

(c) Vocal resonance.

Unaltered.

2. *Chronic bronchitis and emphysema.*

(a) Breath sounds.

Quality—vesicular.

Intensity—normal, weak.

Rhythm—expiration prolonged.

(b) Adventitious sounds.

Rhonchi—sonorous or sibilant.

Râles—bubbling (coarse, medium, fine).

Friction (Lænnec).

(c) Vocal resonance.

Unaltered.

3. *Bronchiectasis.*

(a) Breath sounds.

Quality—vaguely blowing, cavernous, amphoric, cog-wheel.

Intensity—variable.

Rhythm—expiration may be prolonged.

(b) Adventitious sounds.

Rhonchi.

Râles—bubbling, crepitant, gurgling, croaking.

(c) Vocal resonance.

Bronchophony.

Pectoriloquy.

4. *Bronchial asthma.*

(a) Breath sounds.

Quality—vesicular.

Intensity—normal, weak.

Rhythm—expiration prolonged.

(b) Adventitious sounds.

Rhonchi.

Râles—bubbling.

(c) Vocal resonance.

Unaltered.

5. *Plastic bronchitis.*

Auscultatory signs cannot be distinguished from those of acute and chronic bronchitis.

6. *Pneumonia.*

(a) Breath sounds.

Quality—normal, bronchial, tubular.

Intensity—normal, harsh, weak, suppressed.

Rhythm—normal, expiration prolonged.

(b) Adventitious sounds.

Friction.

Rhonchi.

Râles—crepitant, bubbling.

- (c) Vocal resonance.
 - Bronchophony.
 - Pectoriloquy.
 - Diminished resonance.

7. *Broncho-pneumonia.*

- (a) Breath sounds.
 - Quality—normal, faintly bronchial, bronchial.
 - Intensity—normal, harsh, weak, suppressed.
 - Rhythm—expiration may be prolonged.
- (b) Adventitious sounds.
 - Rhonchi, sibilant.
 - Râles—fine crepitant.
- (c) Vocal resonance.
 - Increased.
 - Diminished.
 - Unaltered.

8. *Passive congestion and œdema.*

- (a) Breath sounds.
 - Quality—vesicular, feebly blowing.
 - Intensity—weak.
 - Rhythm—expiration may be prolonged.
- (b) Adventitious sounds.
 - Râles—crepitant, fine, bubbling.
 - Rhonchi.
- (c) Vocal resonance.
 - Unaltered.
 - Slightly increased.

9. *Cirrhosis of lungs.*

- (a) Breath sounds.
 - Quality—cavernous, amphoric, bronchial.
 - Intensity—variable.
 - Rhythm—expiration may be prolonged.

(b) Adventitious sounds.

Râles—bubbling, crepitant.

(c) Vocal resonance.

Increased.

10. *Carcinoma of lungs.*

(a) Breath sounds.

Quality—vesicular, bronchial.

Intensity—variable.

Rhythm—expiration may be prolonged.

(b) Adventitious sounds.

Rhonchi.

Râles.

(c) Vocal resonance.

Normal.

Increased.

Diminished.

11. *Tuberculosis.*

(a) Breath sounds.

Quality—vesicular, bronchial, tubular, cavernous.

Intensity—harsh, normal, weak.

Rhythm—expiration prolonged, normal.

(b) Adventitious sounds.

Rhonchi.

Râles—bubbling crepitant, fine, medium, coarse, moist, dry.

(c) Vocal resonance.

Normal.

Increased.

Diminished.

12. *Pleural effusion.*

(a) Breath sounds.

Quality—vesicular, bronchial, cavernous.

Intensity—normal, weak, suppressed.

Rhythm—expiration may be prolonged.

(b) Adventitious sounds.

Friction.

(c) Vocal resonance.

Normal.

Diminished.

Bronchophony.

Ægophony.

13. *Pneumothorax.*

(a) Breath sounds.

Quality—amphoric, tubular.

Intensity—weak, increased.

Rhythm—expiration prolonged.

(b) Adventitious sounds.

Amphoric hum.

Metallic tinkling.

Bruit d'airain.

Succussion splash.

Râles.

(c) Vocal resonance.

Diminished.

LECTURE V.

PLEURISY.

SUMMARY.

Causes of pleurisy, relation of tuberculosis to pleurisy, clinical experience on this point, bacteriology, inoculation experiments, trials of tuberculin for diagnostic purposes in pleurisy, influence of chill, statistics, influence of the infective fevers and constitutional diseases.

Diagnosis of pleurisy—difficulties in connection with the recognition of pleural friction and of pleural effusion, signs of pleural effusion, bronchial breathing and cavernous breathing in pleural effusion, differential diagnosis of pleurisy and pneumonia at an early stage, pleuro-pneumonia.

Prognosis in pleurisy.

Treatment of pleurisy—general treatment, the relief of pain, promotion of absorption of the fluid, indications for tapping, contraindications, mode of tapping, dangers of tapping, results of tapping.

Chronic pleurisy—problems of treatment in connection with.

Empyema—diagnosis, treatment.

I PROPOSE in the present lecture to give a short account of the subject of pleurisy—especially from the point of view of diagnosis and treatment—dwelling chiefly on those points where difficulties and differences of opinion are likely to arise.

First, as regards cause, pleurisy may be due to—

(a) Chill. In a series of 74 cases under my own observation there was a history of chill in 26.* It is, of course, open to argument that the history may have been in

* Article, Diseases of the Pleura, *Encyclopædia Medica*, vol. 9.

certain cases erroneous. There is the further question whether, granting the correctness of the history, chill was a predisposing or an exciting cause. We may take it, however, that a definite history of chill, *e.g.* a specific wetting, lying on wet grass, rapid cooling when overheated, may be obtained in about one-third of the cases of acute pleurisy which arise in practice.

(*b*) Influence of co-existing diseases, *e.g.* pneumonia, pericarditis, peritonitis, Bright's disease, septicæmia, scarlatina, small-pox, cancer.

Pleurisy is not common in typhoid fever or influenza.

There is no sufficient ground for recognizing the existence of a "rheumatic" type of pleurisy.

I reserve the case of tuberculosis for separate consideration.

(*c*) Traumatic causes.

Relation of Pleurisy and Tuberculosis.

This is one of the most important outstanding questions of practical medicine.

It is now generally admitted that the relation of pleurisy and tuberculosis is much more intimate than was formerly supposed.

The belief that a large portion of pleuritis are tubercular in origin rests upon the following chain of evidence :—

(*a*) Clinical evidence.

(*b*) Bacteriological evidence.

(*c*) Evidence of morbid anatomy.

(*d*) Experiments with tuberculin.

(*a*) The *clinical evidence* is as follows :—

A large number of observers, amongst whom may be mentioned Fiedler, Barrs, Bowditch, Lemoine, Germain Sée, Coston, Dubville, Landouzy, Kelsch, Vaillard, and Schlenker, maintain that of patients who have passed

through an attack of acute pleurisy a large proportion (from a third to a half) subsequently develop phthisis. On the other hand, a history of pleurisy is one of the commonest incidents in the records of cases of phthisis.

(b) The *bacteriological evidence* turns mainly upon the results of inoculation experiments.

The bacillus of tubercle is rarely present in pleural effusions. According to Mitchell Prudden, Netter, Aschoff, Lemoine, and many other observers, a large proportion of cases of pleural effusion are sterile, and the opinion has gained ground that "sterile" effusions are significant of tuberculosis. The proportion of sterile—and hypothetically tubercular—effusions is put by some of these observers at 70 per cent. and upwards.

Inoculation experiments with "sterile" effusions have, in a considerable proportion of cases, produced tuberculosis.

(c) *Evidence of morbid anatomy.*

In fatal cases of pleurisy tubercle is often found in the pleura, the lungs, and the bronchial and retro-bronchial glands. The statistics on this subject are somewhat conflicting, but it may be safely taken for granted that in a large proportion of fatal cases of pleurisy the presence of tubercle can be demonstrated.

(d) *Evidence from the use of tuberculin.*

Koch found that 73·2 per cent. of cases of pleurisy reacted to tuberculin.*

It would be beyond the scope of these lectures to enter into these various questions in detail, or to give a complete synopsis of the statistical evidence at present available. I must content myself with a few general criticisms.

That a very large proportion of fatal cases of pleurisy are of tubercular origin may be admitted. These fatal

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 95.

cases, however, constitute only a small proportion (about 6 or 7 per cent.) of the total.

Much more important questions are the following :—

- (a) What ground is there for supposing that an attack of acute pleurisy, from which recovery is, or seems to be, complete, was tubercular in origin?
- (b) If a patient has made an apparently good recovery from an attack of acute pleurisy, is the danger of subsequent phthisis slight or serious?
- (c) If a patient has made an imperfect recovery from an attack of acute pleurisy, is the danger of subsequent phthisis extremely grave, or only moderately serious?

These questions are of great practical importance. They meet us constantly in practice, they embarrass our forecasts in many pulmonary conditions, they complicate the problems of life assurance. Their solution would be welcome. At present a good deal of doubt hangs over the whole subject.

While granting most fully that the relation of pleurisy and tuberculosis is close, I wish to suggest a few considerations which may make us pause before assenting to the extreme view on this subject which at present prevails especially in France, where medical opinion in a large measure endorses Germain Sée's dictum that "so-called simple pleurisy from a chill is only a tuberculous pleurisy, the nature of which has been misunderstood."

We may ask—

- (a) Is the clinical evidence of the tubercular origin of many acute pleurisies really as strong as it looks?

A patient passes through an attack of acute pleurisy, makes a good recovery, and in one, two, or five or ten years dies of phthisis. What assurance have we that the onset of phthisis was causally related to the previous

attack of pleurisy? May it not have arisen independently? The answer would, no doubt, be that the relation of phthisis to pre-existing pleurisy is too close to be fortuitous. But, it may be rejoined, is it not possible that a non-tubercular pleurisy may in some way lower the general health or weaken the resisting power of the lungs so as to render them more liable to bacillary invasion? This possibility must be admitted.

When phthisis follows pleurisy it is clear that there are three tenable views—

1. That the relation of the two was accidental.
2. That the pre-existing pleurisy was non-tubercular in character, but that it in some way rendered the lungs more vulnerable to bacillary attack.
3. That the pre-existing pleurisy was tubercular.

Writers on the subject have not always weighed these alternatives carefully.

We may ask—

(*b*) Is the clinical course and event of acute pleurisy in ordinary cases suggestive of a tubercular causation? A cautious answer should, no doubt, be given to this question, because our knowledge of tubercular processes is not complete, and we might arbitrarily assume that certain symptoms and a certain type of clinical course were inconsistent with a tubercular causation, while a wider knowledge might show that there was no real inconsistency.

It will be admitted, however, that if ordinary acute pleurisies are in a large majority of cases tubercular in origin, we shall be obliged to revise our conceptions of tuberculosis of serous membranes.

Pleurisy has in most cases a definite onset—chill of some sort is a frequent antecedent; its course is acute or subacute (more often, I think, the latter); the temperature, though not very characteristic, is unlike that of tubercu-

losis ; in a very large majority of cases the disease ends in apparently complete recovery in from three to four weeks ; there are no definite sequelæ.

Now, if we compare this picture with that of, *e.g.*, tubercular meningitis or tubercular peritonitis, the resemblances are not close, the points of contrast are many. This argument is not final, for tubercular pleurisy may chance to be beyond comparison more benign than tuberculosis of the meninges or the peritoneum. But we should have expected more points of resemblance.

If, bearing in mind what is certainly known of tuberculosis, we ask the question, Is it inherently probable that a tuberculosis of a serous membrane will come on acutely or subacutely in a previously healthy individual, run a fairly defined course of three or four weeks, end usually in complete convalescence without obvious impairment of the general health, and then in one, two, or more years burst into fresh activity and involve the lung parenchyma? the answer is not easy.

Without dogmatizing on this difficult subject, it will, I think, be admitted that from the clinical side there is a good deal to be said for the view that a pleurisy, not itself tubercular, may prepare the ground for tubercular invasion of the lungs.

The evidence derivable from morbid anatomy does not touch the special question just now under consideration.

The results of inoculation experiments, though in the main corroborative of the theory of the close connection of pleurisy and tuberculosis, are not uniform, and are open to the fallacy that what is true of the guinea pig is not necessarily true of man.

Koch's experiments with tuberculin in cases of pleurisy are important, and lend support to the view that the majority of cases of acute pleurisy are of tubercular causation.

In this connection, however, we might do well to reflect upon all that is involved in the well-known dictum, *Jedermann hat am Ende ein Bischen Tuberkulose*.

The whole question thus remains in a somewhat ambiguous position. I would suggest the following tentative conclusions :—

- (a) That the connection of pleurisy and tuberculosis is much more intimate than was formerly supposed.
- (b) That the view that the great majority of acute pleurisies are of tubercular origin does not rest upon any secure pathological basis, and is not easily reconciled with the clinical facts.
- (c) That an attack of acute pleurisy occurring in a previously healthy individual, and completely recovered from, is not a sufficient ground for apprehending the onset of pulmonary tuberculosis provided the habits and mode of life are favourable to health.
- (d) That such cases may be accepted for life policies at ordinary rates, if two years have elapsed since the attack of pleurisy, and if the general health, pulmonary expansion, and weight are satisfactory.
- (e) That if an attack of pleurisy have left behind an unexpanded lung or impaired general health, the probability of subsequent phthisis is considerable. Quite a large number of these cases, however, remain short-winded and debilitated, but do not become tubercular.

In concluding our survey of this subject, we must not forget to remark that acute pleurisy may supervene in patients who have a quiescent tubercular lesion at one or both apices. This is probably a not uncommon event. Patients suffering from pleurisy often give a history of previously indifferent health, and especially of a "weak

chest." In such cases, it is well to weigh the probability of pre-existing phthisis.

Diagnosis of Pleurisy.

The diagnosis of pleurisy is in the large majority of cases an easy matter. It rests essentially upon physical signs, and only in a minor degree upon symptoms and history. Pleural friction and the signs of effusion are in most cases unequivocal. Difficulties, however, sometimes arise. I shall consider them from two points of view.

(a) Difficulties in connection with the recognition of pleural friction.

(b) Difficulties in connection with the recognition of pleural effusion.

(a) Difficulties in connection with the recognition of pleural friction.

Pleural friction has usually the following characters: It is rubbing or scraping (exceptionally crackling or crepitant) in character. It is "superficial." It is heard both during inspiration and expiration. It is increased in intensity by a deep inspiration. It is not affected by coughing, either as regards its intensity or its area of distribution. It is often accompanied by friction fremitus and by localized pain. It is usually confined to a small portion of the chest, but exceptionally may be heard over a wide area. Its most usual seats are the inferior antero-lateral and posterior regions of the chest. It is entirely annulled when the breath is held, unless the slip of lung over the pericardium is involved. In this latter case it may be excited or modified by the movements of the heart.

The chief difficulty of diagnosis arises in such a case as the following: The symptoms and history suggest pleurisy or pneumonia. The only physical sign is a sound at one base which may be either crepitus or crepitant friction.

It will be remembered that many observers now regard the so-called "primary crepitus" of pneumonia as really due to pleural friction. Excluding this condition, which can only give rise to temporary difficulty, what considerations will assist us in distinguishing in a doubtful case between friction and crepitus? I suggest the following rules:—

1. Pleural friction is more "superficial" than crepitus. The term "superficial," as applied to a sound in the lungs, involves, of course, both an observation and an inference. We cannot directly observe the "superficiality" of a sound. What we mean by the term is this—I hear a sound of a certain quality, and from that quality I infer that the sound is produced on the surface of the lung, and not in the ramifications of the bronchioles. It is obvious that error may lurk either in the observation or the inference. Yet the point is an important one, and by close attention to the quality of the sound I believe we may often determine with accuracy that it is "superficial," and hence pleural.

2. Pleural friction is, with few exceptions, audible both during inspiration and expiration. Crepitus is frequently confined to inspiration, especially the end of inspiration. There are exceptions to this rule, *i.e.* friction may be limited to inspiration (this is rare), and crepitus may be heard during both inspiration and expiration (this is not uncommon); but, upon the whole, the rule is a useful and generally trustworthy one. A sound confined to the end of inspiration is probably crepitus.

3. Pleural friction is not altered in quality or area of distribution by coughing. Crepitus is frequently altered by coughing, both as regards quality and area.

4. Pleural friction is usually accompanied by local pain—crepitus is not.

5. Pleural friction is often intensified by pressure of the stethoscope—crepitus never.

6. Examination of the chest in the neighbourhood of the doubtful sound may reveal unambiguous friction or crepitus.

Pericardial friction is usually distinguished without much difficulty from pleural friction. It is much more rapid than pleural friction. It has a peculiar "to-and-fro" quality. It is confined to the cardiac area. It is definitely related to the movements of the heart. It is independent of the respiratory movements. It is usually loudest when the breath is held after a deep inspiration. It is louder when the patient sits up or leans forward than in the recumbent position.

From all these points of view, pericardial friction presents a contrast with pleural friction.

(b) Difficulties in connection with the recognition of pleural effusion.

These difficulties are in certain exceptional cases great, even insuperable. The best observers are agreed that the signs of pleural effusion and of pulmonary consolidation may sometimes be indistinguishable. This is, however, rare.

The ordinary physical signs of pleural effusion are as follows :—

1. Inspection.

The affected side is more or less immobile. It may be more prominent than the sound side. It may be obviously larger than the sound side. The intercostal spaces are usually (not always) flattened. Bulging of the spaces is excessively rare. The cardiac impulse is displaced towards the sound side. In certain very rare cases visible pulsation may be seen over the area of the effusion. The excursion of the diaphragm is limited.

2. *Palpation.*

Vocal fremitus is in most cases diminished or absent. Exceptions to this rule occur—probably due to pleural adhesions.

The position of the cardiac impulse may be further defined by palpation.

3. *Percussion.*

Dulness is present, often wooden in quality.

Dulness increases in intensity towards the base.

The outline of the dulness is often characteristic. The highest point of the dull area is usually posterior—sometimes in the axilla. Ellis's curve may be well marked. The outline of the dull area is not usually affected by changes of posture on the part of the patient. In passive effusions the rule is the contrary.

Skodaic resonance is often elicited under the clavicle upon the affected side.

Exceptionally, the cracked-pot sound may be audible above the dull area.

The sense of resistance is much increased.

4. *Auscultation.*

The breath sounds are usually weakened, often annulled. Bronchial breathing is common in the inter-scapular region on the affected side. It is occasionally heard over the whole of the affected side, especially in children. Cavernous breathing is sometimes present. In cases where there is a thin layer of fluid the breathing may be fairly well marked and vesicular in quality.

There are no adventitious sounds until absorption has begun, when friction is usually audible. At this stage, crepitant râles are sometimes present.

Vocal resonance is usually reduced. In certain cases, owing to adhesions, it may be increased, and even bronchophony may be present.

Ægophony is occasionally audible above the level of the effusion posteriorly in the region of the scapula.

In some cases of pleural effusion the voice seems simply flat and devoid of musical quality, rather than weakened.

5. *Mensuration.*

The affected side often measures more than the sound side, but exceptions to this rule are not uncommon.*

Errors in diagnosis between pleural effusion and pneumonic consolidation arise chiefly by misinterpretation of the auscultatory signs and inattention to the position of the cardiac impulse.

It is not always remembered—

(a) That bronchial breathing is common in effusion.

(b) That weak or suppressed breathing is common in pneumonia—chiefly, probably, in connection with blocking of the bronchial tubes by secretion.

The bronchial breathing of pleural effusion cannot in all cases be definitely distinguished from that present in pneumonic consolidation. As a rule, however, it is weaker, more “distant,” less often loud and definitely tubular than the breathing of pneumonia.†

Consolidated lung, with blocking of the tubes by secretion, may give rise to a group of physical signs hardly to be distinguished from those of pleural effusion. In such cases we find dulness on percussion, loss of vocal fremitus, absence of breath sounds, diminution of vocal resonance. The position of the cardiac impulse is here the best touchstone of diagnosis, while the history of the case, the character of the sputum, attention to the outline of the dulness, and the fact that blocking of the tubes is only a temporary phenomenon in pneumonia, will usually keep us right.

* See *ante*, p. 77.

† See *ante*, p. 67.

Malignant disease of the lungs may give rise to physical signs closely simulating those of pleural effusion. The history and symptoms in the two cases are usually different.

The state of the vocal fremitus is always a point of great importance where the diagnosis of pleural effusion is in question. Increased intensity of vocal fremitus is strongly suggestive of consolidation. Exceptions to this rule occur under the same circumstances as weakening or absence of the breath sounds. The fremitus is very rarely increased in effusion. Diminution of the vocal fremitus must be interpreted with some caution. It is the rule in pleural effusion, but it is not uncommon, as a temporary condition, in pneumonia, when the tubes are blocked by secretion.

Gee has given a good account of certain cases in which the physical signs of pleural effusion simulate those of tubercular excavation of the lungs. Several cases of this kind have occurred in my experience. As this point is not usually much dwelt on, I will state Gee's views in full : "At places the breath sounds (*i.e.* in pleural effusion) may be bronchial in all degrees of intensity, up to a hollow resonance, such as Lænnec himself would have supposed to leave no doubt of cavity. Add to the physical signs hectic fever, and we cease to wonder that pleurisy of this kind is usually mistaken for phthisis more or less advanced. However, the pleuritic patients recover completely, without a vestige of disease left behind, save, haply, a slight unilateral retraction of the chest, or a cup-like depression. Whenever the signs of a supposed phthisis are in some respects peculiar, whenever they indicate advanced disease limited to one side of the chest, whenever cavernous signs are heard in unusual places, it is well to weigh the possibility of simple pleurisy. The most useful guide to

physical diagnosis is this—that, as a rule, the signs of pleurisy are more marked in the lowermost part of the chest, and that the signs of phthisis are more marked at the upper part.”*

I will add two comments on these cases, which I believe are not excessively rare. Gurgling is not heard in the cases of pleurisy, and attention to the position of the cardiac impulse may give important assistance.

The following diagnostic problem is common in practice. A patient is seen on the second or third day of a severe illness, characterized by sharp pyrexia, dyspnœa, and thoracic pain. The physical signs, for some of the reasons already adduced, are ambiguous. How shall we decide whether we are dealing with commencing pleurisy or commencing pneumonia, other alternatives being excluded?

I suggest the following aids to diagnosis in such cases:—

1. A sudden onset may occur in either condition, but is much commoner in pneumonia.
2. A gradual onset, extending over three or four days, is not unknown in pneumonia, but is excessively rare in this disease, while it is common in pleurisy.
3. A single severe initial rigor is strong presumptive evidence of pneumonia.
4. Severe pain in the chest has no weight, being usual in both diseases.
5. Labial herpes suggests pneumonia, but is not to be relied on.
6. Marked alteration of the pulse-respiration ratio is strong evidence of pneumonia.
7. The amount of physical prostration is much more

* Auscultation and Percussion, third edition, p. 221.

marked in pneumonia than in pleurisy. The pneumonic patient often lies like a log in bed, and raises himself with difficulty for examination. The pleuritic patient is often restless and anxious, but not prostrate.

8. The type of dyspnœa present in the two cases is different. The *besoin de respirer* is much more marked in pneumonia than in pleurisy. The pneumonic patient does not take a deep breath, because he cannot. The pleuritic patient does not take a deep breath, because he is afraid of the pain.

9. Mental symptoms—delirium, excitement, stupor—are much commoner in pneumonia than in pleurisy.

10. The tongue in early pneumonia commonly presents a thick creamy fur. This is not characteristic of pleurisy.

11. The urine in pneumonia is deficient in chlorides and often contains albumen. We do not find these characters in the urine of pleurisy.

12. The sputum in pneumonia, if present, is viscid and rusty in most cases, sometimes sanguineous, sometimes like prune-juice. The sputum of pleurisy is catarrhal in character.

We must bear in mind in this connection—

(a) That some degree of pleurisy is invariable in pneumonia.

(b) That genuine pleuro-pneumonia, which is common in cattle, is rare in man.

The diagnosis of pleuro-pneumonia is usually the resource of the weak diagnostician.

On the whole, I have more often suspected pneumonia where the subsequent development proved the case to be pleurisy than *vice versa*. Sthenic cases of pleurisy in young people are apt to be misleading.

I suggest the following rules:—

(a) In presence of a case which I suspect to be pneumonia, but recognize may be pleurisy, watch the cardiac impulse, weigh symptoms.

(b) In presence of a case which I suspect to be pleurisy, but recognize may be pneumonia, watch the vocal fremitus and vocal resonance, note the pulse-respiration rate, ask for the sputum, examine the urine.

(c) Do not give a diagnosis of pleuro-pneumonia. It is usually a wrong diagnosis. In the great majority of cases the disease is either pneumonia or pleurisy, not a hybrid.

The suspicion of existing phthisis sometimes arises in the course of pleural effusion. West speaks as follows on this subject: "If the lung be adherent at the apex, the physical signs may become puzzling. Thus the breath sounds may be bronchial, there may be bronchophony, and occasional crepitation. It will then be impossible to say that the patient is not suffering from phthisis, and it will be necessary to wait till the fluid has disappeared before this question can be settled. In a very large number of cases all these suspicious signs disappear when the fluid has been removed, and no evidence of apex mischief remains." *

This account of these cases is quite in accord with my own experience.

It is a good clinical rule not to give an opinion as to the existence of phthisis during the continuance of pleural effusion. The nature of the fluid does not give us any decisive assistance. A sanguineous effusion lends some support to the suspicion of tubercle, but a serous effusion does not throw any light on the differential diagnosis. A purulent effusion suggests pneumococcal

* Diseases of the Organs of Respiration, S. West, vol. ii. p. 664,

invasion, rather than tubercle. Bacteriological examination of the fluid does not usually afford the basis for diagnosis.

Pleurisy without exudation may be mistaken for any of the following conditions :—

Pleurodynia.

Intercostal neuralgia.

Periostitis of one or more ribs.

The differential diagnosis in these cases is not usually difficult. In pleurodynia the muscles are tender to the touch, movement is acutely painful, the temperature is normal, and there are no auscultatory signs. In intercostal neuralgia, a careful analysis of the seat and distribution of pain will prevent error. In periostitis of a rib, local tenderness and the absence of any of the auscultatory or general symptoms of pleurisy will assist the diagnosis.

Prognosis in Pleurisy.

On this subject we may lay down the following rules :—

- (a) The dangers of the acute stage are, upon the whole, slight, but sudden death during this stage is not unknown.
- (b) Tedious convalescence is common, and may either lead to phthisis or impair the general health in other ways.
- (c) Lingering cases not uncommonly ultimately make a good recovery.
- (d) Phthisis may ultimately ensue, even when recovery has appeared complete.
- (e) Serous effusions do not often become purulent.
- (f) A slight degree of dulness and weakened breathing at the base of the lung after pleurisy, provided

the general health is satisfactory, is of no consequence.

Our attitude towards tedious cases of pleurisy is a difficult one, the chance of tuberculosis being always before our minds. It is not well to assume that every intractable case of pleurisy is on the road to phthisis. Quite a considerable number of these cases make a good recovery. Much depends upon the patient's occupation and mode of life, and the prospects of rational treatment.

Treatment of Pleurisy.

In the initial stage of pleurisy the relief of pain is the chief indication. Morphia or opium in some form is the obvious remedy. A tight bandage round the chest is relieving to the patient. Roberts recommends fixing the chest by strips of plaster.

When effusion has set in the treatment consists in febrifuge measures and gently stimulating the emunctory organs. Such remedies as citrate of potash, bitartrate of potash, and acetate of ammonia may be given. The value of local counter-irritation is not assured, but such measures are usually employed. As the great majority of pleural effusions are spontaneously absorbed, there is no need for the display of therapeutic activity at this stage. Severe purgation and active diaphoresis are usually unnecessary, and possibly hurtful.

When the pyrexia has subsided, usually towards the end of the second week, one of the best combinations is a mixture containing iodide of potash and citrate of iron and ammonia.

The diet should be simple and light, but the severe limitation of fluids, with the view of promoting absorption, is not to be recommended.

If absorption is delayed, *i.e.* if no progress is manifest

by the beginning or middle of the third week, it is probable that the case belongs to the less favourable type, viz. that in which spontaneous absorption does not occur. The choice now lies between active saline purgation and vigorous stimulation of the skin and kidneys on the one hand, and tapping on the other. I have no hesitation in decidedly preferring the second alternative. It is easy, safe, prompt, and efficient, while the former measures are of doubtful efficacy and tend to weaken the patient in a condition when—in view of the possibility of consecutive tuberculosis—all debilitating methods are undesirable. I have seen cases where, in spite of drenching perspirations and profuse purgation, the effusion remained absolutely stationary.

Tapping, with or without aspiration, is a modern procedure, and it is well sometimes to ask ourselves the question, What became of pleural effusions in the days before tapping? Undoubtedly the great majority of them were absorbed. Nevertheless, tapping—in suitable cases, and performed with due precautions—is a measure of the highest utility. It is immediately effective in a certain proportion of cases, while it starts other cases on the road to recovery. It is usually free from serious risk. It is an operation which does not usually involve any difficulty.

The following rules may be laid down as indications for tapping :—

- (a) Tap if there are no signs of commencing absorption by the middle or end of the third week.
- (b) Tap if there are any symptoms of immediate urgency, *e.g.* marked dyspnœa, interference with the action of the heart, cyanosis.
- (c) Tap if the fluid reaches to the second interspace in front.

(*d*) Tap if the effusion is double.

There are no definite contra-indications as regards tapping. Fever is not a contra-indication, nor extreme weakness of the patient, nor old age, nor the chronicity of the case, nor the probable presence of tubercle, nor any peculiarities of the effusion. These points will all require consideration in a doubtful case, but they must not be held definitely to contra-indicate tapping. We shall reconsider these points later on.

It is well to perform a preliminary exploration before tapping, in order to establish the diagnosis, and to obtain a specimen of the fluid. The ordinary hypodermic needle is useless for exploring the chest. A negative result obtained by its employment is no evidence of the absence of fluid. A special exploring syringe, now made by several of the leading instrument manufacturers, should be employed, and great care taken to secure that it draws well. Where the physical signs clearly indicate the presence of fluid, a negative result of exploration must be cautiously interpreted. Failure to find fluid may be due to its loculation, or to pushing the pleura before the needle, or to blocking of the needle by exudate, and to other causes.

The indications for tapping, above enumerated, do not call for any detailed comment.

The time limit suggested is, of course, an arbitrary one, but we must have some approximate rule. There are good grounds for believing that tapping in the first week of pleural effusion is undesirable. It seems to stimulate the activity of the disease, and to render a recurrence of the effusion certain. During the course of the second week the propriety of tapping may be considered, but in the absence of active symptoms we shall usually do well to stay our hand. There is always a good chance that

spontaneous absorption may set in at this stage. Our guiding principle is (in the absence of urgent symptoms) to wait until the effusion has attained its maximum, and until there are grounds for thinking that spontaneous absorption is improbable.

The second rule is imperative. If urgent symptoms set in where effusion exists, tapping should be promptly performed.

The third rule, like the first, is obviously somewhat arbitrary. Yet the amount of fluid is a point of real importance in weighing the propriety of tapping. Small effusions are certainly more prone to spontaneous absorption than large effusions. Where the effusion quite, or almost, fills the side, it is usually inadvisable to wait for spontaneous absorption. Some authorities favour the tapping of small effusions.

The fourth rule is obvious, and does not call for comment.

I have said that there are no definite contra-indications as regards tapping. The suggestion that the presence of pyrexia is a contra-indication is certainly unfounded. In pyrexial cases a fall of temperature as the consequence of tapping is common. Extreme weakness of the patient is not a contra-indication. In such cases the possibility of benefit from the operation outweighs its risks. Old age is not a contra-indication. I have performed aspiration with good results in a patient aged 87. The probable presence of tubercle can no longer be held as a contra-indication, in view of the probability that a large proportion of cases of apparently simple pleurisy are really tubercular in origin. The chronicity of the case does not preclude tapping. West has seen complete re-expansion of the lung in a case of effusion of eighteen months' duration.*

* Diseases of the Organs of Respiration, S. West, vol. ii. p. 685.

The question of the light thrown upon the propriety of tapping by the nature of the fluid is an important one.

If the fluid be serous, tapping, if otherwise indicated, may proceed. If the fluid be purulent, tapping will probably be ineffectual, but may be performed either as a preliminary to free incision and drainage or (less desirably) as a substitute for those measures, if for any reason they are not undertaken. If the fluid be bloody, the question of tapping requires much consideration. A bloody effusion may be found in the following conditions: tuberculosis, cancer, granular kidney, cirrhosis of the liver, hæmophilia, purpura, malignant fevers, pulmonary infarction. It is evident that in these conditions the question of tapping is complicated by so many other considerations that no definite rules can be laid down. As a broad rule, tapping should be performed very cautiously, if at all, in bloody effusions. Often it is useless, sometimes it is probably dangerous. I have tapped several cases of bloody effusion. In one case, which was probably tubercular, the result was beneficial, in the others doubtful.

Mode of performing the operation.

Aspiration is not always necessary. A trocar and evacuating tube are sometimes sufficient. If aspiration be decided upon, either Dieulafoy's or Potain's aspirator may be employed. In careful hands both of these are excellent instruments, but I am disposed to give the preference to that of Dieulafoy, inasmuch as, though somewhat more difficult to manage, it permits of a readier control of the amount of vacuum than the instrument of Potain. Strict antiseptic precautions should be taken. The entrance of air into the pleural cavity cannot always be prevented, but does not usually lead to any serious consequences. A large or

medium-sized trocar or needle should be employed. A small trocar or needle often leads to difficulties, especially in dealing with thick effusions, and in our desire to spare the patient a very slight additional pain we may hamper ourselves unnecessarily, and ultimately put the patient to far more serious inconvenience.

Place of puncture.

The points of election for puncture are either—

- (a) The mid-axillary line in the fifth or the sixth space on the right side, and from the fifth to seventh space on the left.
- (b) Below the inferior angle of the scapula in the eighth space.

There is no advantage in puncturing the most dependent part, as syphonage renders the operation independent of gravity.

The advantages of the former site are as follows:—

- (a) The chest wall is easily pierced.
- (b) The pleura is not usually much thickened.
- (c) The ribs are not pressed down upon each other, and there is usually ample room.

The disadvantages of this site are as follows:—

- (a) There is some (not much) danger of wounding important vessels.
- (b) There is a possibility of injuring the heart or liver.

This danger is not serious.

The advantages of the latter site (below the inferior angle of the scapula) are as follows:—

- (a) It is remote from important vessels and viscera.
- (b) It facilitates the assumption of the correct posture by the patient.
- (c) It prevents the patient from observing the details of the operation.

The disadvantages of this site are as follows:—

- (a) The chest-wall may be difficult to pierce, owing to muscle or fat.
- (b) The pleura is often much thickened at this point,
- (c) The ribs are sometimes pressed down the one upon the other in this situation, so that there is a difficulty in effecting an entrance.

Upon the whole, I prefer the posterior site.

Dangers of tapping.

These are of two kinds, viz.—

- (a) Dangers arising during the performance of the operation.
- (b) Dangers arising subsequent to the performance of the operation.

(a) Dangers arising during the performance of the operation.

The chief of these is syncope. A slight degree of syncope is not uncommon. Severe syncope has, however, been very rare in my experience.

Syncope may be averted by attention to the following points:—

1. *The posture of the patient.*—The patient should on no account be allowed to sit up during tapping. He should be semi-recumbent, with the shoulders and head well propped up with pillows, and with the affected side turned towards the operator.

2. If the pulse be weak, a little alcohol may be given before tapping, and $\frac{1}{20}$ grain of strychnine may be injected hypodermically. Digitalis and strophanthus are too slow in their action to be of service in this connection.

3. Anæsthesia—either local or general—should be

avoided. It is unnecessary, and not free from risk. The amount of pain inflicted by the operation is not serious.

4. If syncope threatens, we should not attempt to empty the chest, but rest satisfied with a moderate aspiration. When the operation is well borne, there is no objection to continuing aspiration so long as the fluid flows freely.

Coughing sometimes arises during tapping. The patient should be instructed to restrain it as much as possible, and may take a little brandy. If the coughing is severe the operation should be temporarily suspended, and if it prove uncontrollable (which is unusual) tapping should be abandoned.

Severe pain in the side is often complained of, and has various causes. Wound of the lung is not the most frequent cause. As a general rule, pain, unless very severe, is not a sufficient cause for abandoning the operation.

Hæmorrhage may supervene during tapping. It is most often due to rupture of vessels in the wall of the pleura, in consequence of being suddenly deprived of their usual support. In such cases the operation should be abandoned. Hæmorrhage hardly ever results from a wound to the intercostal artery. The case of bloody pleural effusion has been previously considered.

(b) Dangers subsequent to tapping.

The most grave of these dangers is acute œdema of the lung, which may set in within a few minutes or hours after tapping.

Its onset is characterized by intense dyspnœa, cyanosis, faintness, rapid weak irregular pulse, and the expectoration of a large quantity of watery fluid. The

physical signs are fine moist crepiti over the affected area, with perhaps a slight diminution of percussion resonance. The condition is a very grave one, and usually ends fatally. Alcohol, strychnine, the nitrites, oxygen, and dry cupping may be tried. The propriety of venesection may be considered. Acute œdema of the lung is fortunately a very rare sequel of tapping.

Upon the whole, the above accidents in connection with tapping are quite exceptional.

Treatment of Chronic Pleurisies.

Two types of case may be recognized:—

(a) The case where, after pleurisy, there is much retraction of the lung, with little or no residual effusion. In such cases the chest-wall falls in, the heart is much displaced, the patient is permanently short of breath, and perhaps more or less debilitated.

The treatment in such cases consists in respiratory gymnastics, change to upland or mountain air, and attention to the general health and nutrition.

(b) *Cases of recurring effusion.*—In certain cases the effusion persistently recurs after numerous tapplings. Many of these cases are probably tubercular. In such cases we have to decide amongst the following courses:—

(1) To continue the tapplings.

(2) To incise and drain the chest, according to the method employed so successfully in empyema.

(3) To try antiseptic injections.

(4) To rely upon change of air and hygienic measures.

The first and the third of these alternatives are probably inadvisable.

The second alternative, viz. incision and drainage, has not been largely tried. It is, however, recommended by Osler and by West.

The fourth plan may always be tried, and sometimes succeeds even in apparently unpromising cases.

I have learnt by experience not to pass an absolutely unfavourable verdict upon even the most rebellious cases of chronic pleurisy, provided the patient's nutrition is fairly good, and there is no evidence of progressive phthisis or of amyloid disease. Some of these very troublesome cases can be coaxed back to a condition of comparative health by an occasional judicious tapping, suitable change of air, and attention to general hygienic measures.

Empyema.

With empyema I shall deal very briefly, as there is substantial agreement on all the main issues.

Two points have been impressed upon me by my own experience :—

(1) The impossibility of distinguishing pus from serum by any evidence short of puncture.

The rules often laid down on this subject are fallacious. Neither signs nor symptoms will definitely serve to distinguish pus from serum. I have known cases where a hectic temperature, profuse sweating, debility, and wasting co-existed with a limpid serous exudation, and *per contra* cases where all these symptoms were absent, and yet pus was found. Marked tenderness of the side and œdema of the skin are certainly commoner in purulent than in serous effusions, but these points are not pathognomonic. Baccelli's sign is, I think, untrustworthy. Where puncture is so easy and so absolutely decisive, it seems foolish to waste argumentation over a point which can be at once determined.

(2) The difficulty in certain cases of finding pus, even when on general grounds we feel confident of its presence.

Loculation, thickened pleura, and inspissated pus which does not flow freely account for these difficulties. In such cases we should not hesitate to make numerous explorations.

The treatment of empyema is a surgical question.

LECTURE VI.

THE EARLY DIAGNOSIS OF PULMONARY PHTHISIS.

SUMMARY.

The meaning of early diagnosis, fallacies upon the subject.

Early diagnosis rests upon—

- A. Family history.
- B. Personal history.
- C. Examination of the sputum.
- D. Symptoms and mode of onset.
- E. Physical signs.

A. Family History.

Proclivity to infection.

B. Personal History.

Occupation ; previous diseases—pleurisy, influenza, whooping cough, measles, typhoid fever ; proneness to “colds upon the chest,” habitual light weight, anæmia, dyspepsia, glandular enlargements.

C. Examination of the Sputum.

Its great importance, fallacies in connection with.

D. Symptoms and Mode of Onset.

E. Physical Signs.

Malformations of the chest, localization of signs, physical signs of early apical catarrh, alterations in the breathing, râles, variability of râles at an early stage of the disease, difficulties as regards percussion, conduction of heart sounds.

Radioscopy.

It is not necessary to dwell upon the great importance of the early diagnosis of pulmonary phthisis. The disease is comparatively tractable in its early manifestations, whereas the fully developed malady is usually incurable. The popular impression that everything depends upon “getting the case in time” is, upon the whole, well founded.

It may be well to inquire what we mean by "early diagnosis."

There was a time when the following would have been regarded as a typical case of incipient phthisis: "The patient has been coughing and losing flesh for weeks or months. There is afternoon pyrexia, and some sweating at night. In the infraclavicular or suprascapular region of one side we can detect slight dulness, some alteration in the quality of the breathing, and a few crepitant or consonating râles. The sputum contains bacilli." It is indispensable to recognize that such a case is not incipient, but advanced, although the disease may be still circumscribed. By the time the patient presents the classical group of symptoms—cough, afternoon pyrexia, wasting—and unequivocal signs at one apex, phthisis has made serious, sometimes irreparable, headway. If at the outset the physician wavers in his diagnosis, temporizes in his treatment, and permits invaluable time to be lost, no subsequent activity and diligence on his part can repair the consequences of his error. The problem of the early diagnosis of phthisis should rather be stated in these forms—What facts or combination of facts as regards history, symptoms, and physical signs afford presumptive evidence that the process of tuberculization has already begun in the lungs? Are we justified in sounding the alarm before we find unequivocal signs in the lungs or bacilli in the sputum? How are we to appraise, on the one hand, suspicious symptoms in the absence of physical signs, or suspicious signs in the absence of symptoms? Is a patient who has had several attacks of hæmoptysis, and who is coughing and losing flesh, to be pronounced tubercular, although we can detect neither definite physical signs in the lungs nor bacilli in the sputum? What are we to say regarding a case where we

find slight dulness and prolonged expiration under one clavicle, while symptoms are practically wanting? These problems might be multiplied indefinitely. They are not fanciful problems. Every physician has felt their weight, and has sometimes wavered in his judgment.

I exclude from consideration all cases where the diagnosis is, with reasonable care and skill, obvious.

It would be easy to say, Do not give a positive diagnosis until either bacilli are found in the sputum or definite physical signs are detected in the lungs. In other cases take refuge in such euphemisms—which the lay mind is only too ready to accept—as “a delicate chest,” “weak lungs,” a “tendency to consumption.” It must be insisted that this is an absolutely disastrous policy. It means in many cases the sacrifice of the patient's best—perhaps his only—chance of recovery. While we are waiting for evidence to relieve us of the risk of diagnostic error, tuberculosis is too often making disastrous progress. Yet, it may be retorted, think of the alarm, inconvenience, and loss which will accrue to our patients if we give a premature diagnosis, if we evoke fears which we may well prove impotent to quell. Precisely, therein lies the crux of the whole question. It is well to begin, however, by realizing that a dilatory policy is fraught with grave peril. Every case where the history, symptoms and signs fairly suggest the possibility of incipient phthisis should be promptly faced alike by physician, patient, and patient's friends. If a positive opinion can be arrived at, then the therapeutic problem arises. If a positive opinion is impossible, it is at least well to have considered the graver alternative. The one thing not to do is to use agreeable euphemisms and quiet alarms which may ultimately prove to have been only too well founded.

We may seek for assistance in the early diagnosis of phthisis by attention to the following points, viz.—

- A. Family history.
- B. Personal history.
- C. Symptoms and onset.
- D. Sputum.
- E. Physical signs, including the results of radioscopy.

A. Family History.

The tendency at the present day is to minimize the importance of this factor. No doubt its significance has been exaggerated in the past. It is a great error, however, to regard it as of no consequence.

I have already expressed my conviction that pulmonary tuberculosis runs more or less in families, the underlying facts being proclivity to infection and special exposure to the virus of the disease. Hence in a doubtful case the history of tubercle in the family is entitled to some weight. We must be careful, however, not to allow such a history to prejudice our opinion in favour of tubercle, nor to permit the absence of such a history to make us unduly sceptical about the probability of tubercle.

A definite family history of phthisis is obtainable in about one-third of all cases.

Members of phthisical families who escape infection are often up to the full normal standard of health.

B. Personal History.

This is much more important than family history. We may consider the following points :—

1. The patient's "constitution," development, weight, and general health history.
2. His occupation.
3. His previous diseases.

4. The presence of anæmia, dyspepsia, glandular enlargements, or scars.

Phthisis rarely arises in persons of healthy aspect, sound digestion, adequate weight, and good blood-making power. Yet even this obvious rule has its exceptions. I can recall at least two cases in which tubercle arose in young men of apparently faultless physique and of quite exceptional vigour and development. But such cases are decidedly rare. Some impairment of general health is a fairly constant forerunner or concomitant of the onset of phthisis.

The shape of the chest will naturally receive attention. The flat chest and the alar or winged chest are common in phthisis. Let it be remembered, however, that the disease not rarely develops in a chest apparently of normal shape and dimensions.

The erethic constitution (*eretische constitution*) is common in connection with phthisis. The condition seems to be one of deficient nervous and vaso-motor stability. The patient is often intellectually alert, responsive to all kinds of stimuli, capable of considerable effort, but soon fatigued, his pulse is too fast, his eye too bright, his skin too moist.

The weight is a point of great importance. Consumptives, in the great majority of cases, show a deficiency of weight both before the outbreak of the disease and during its course. Yet the exceptions to this rule, though not numerous, are important.

A proneness to catarrhs, "colds on the chest," and the like is common in the subjects of tuberculosis. A rule which I find of practical value is this—Does the patient give a history of repeated "colds on the chest"? Then, he is probably either bronchitic or tubercular. If

the former, the chest will tend to be rounded and hyper-resonant; if the latter, the chest will tend to be flat and deficient in resonance.

Occupations involving close confinement in vitiated air involve the heaviest risk of tubercle. Thus the maximum phthisis rate is found amongst such classes as printers, cutlers, file-makers, and earthenware manufacturers, and the minimum rate amongst such classes as agricultural labourers and fishermen.

The fact of an indoor occupation in impure air should make us scrutinize with special care any signs or symptoms suggestive of commencing phthisis.

The diseases most often followed by phthisis are the following: pleurisy, broncho-pneumonia, measles, influenza, whooping-cough, typhoid fever, diabetes, insanity, chronic syphilis, cancer, severe dyspepsia, anæmia.

The relation of pleurisy to phthisis has been already considered. Although some exaggeration has characterized the statements of certain writers who have emphasized the closeness of the relation, yet it would be easy to under-estimate the importance of the connection between the two diseases. Most physicians will endorse Osler's declaration: "I confess that the more carefully I have studied the question, the larger does the proportion appear to be of primary pleurisies of tuberculous origin." *

In one respect pleurisy seems to differ from certain other diseases often followed by phthisis—*e.g.* influenza, measles, whooping-cough—viz. in the fact that even a pleurisy apparently completely recovered from and leaving neither physical signs nor impairment of the general health, seems to involve a legacy of increased proclivity to phthisis. I doubt if this is true of the other diseases

* Practice of Medicine, fourth edition, p. 666.

mentioned, but it seems to be true of pleurisy, however we may explain the fact.

Broncho-pneumonia is followed by phthisis in a not unimportant proportion of cases, while this is never the case, in my opinion, with acute croupous pneumonia. The cases which have seemed to support the theory of a causal link between acute croupous pneumonia and phthisis are probably, without exception, cases of pneumonic phthisis from the outset.

Measles and whooping-cough are often followed by phthisis, especially in ill-nourished and badly-cared-for children. Influenza seems to be a frequent precursor of phthisis. No statement in this connection has been commoner in my experience on the part of the subjects of early phthisis than that they had suffered previously from one or more attacks of influenza. The question arises, Can this history be trusted? Might not the alleged attack or attacks of influenza have been simply among the earlier manifestations of tuberculosis? That this is sometimes true is probable, but the history of genuine influenza, with its sudden onset, severe pains in the head, back, and limbs, profound mental depression, and more or less rapid amendment after the fifth or sixth day, is often too clear to be distrusted. It is well to remember, however, that the term "influenza" is a frequent cloak for diagnostic error, and that the term is sometimes employed to cover those vague chills, feverishness, and debility which mark the earlier developments of tuberculosis.

Typhoid fever is sometimes followed by phthisis, but, bearing in mind how common the disease is, it does not appear that this sequence is a frequent one.

Diabetes is often followed by phthisis. Probably nearly one-half of the subjects of diabetes ultimately succumb to pulmonary tuberculosis.

Insanity seems to predispose to phthisis. Of cases of idiocy and congenital imbecility, two-thirds ultimately die of tubercular disease.* “Consumption was startlingly more frequent as a cause of death among the inmates of the older asylums than in the modern institutions; but still it is in all asylums for the insane between three and four times more common than in the general population at the same ages.”†

Chronic syphilis is frequently associated with phthisis. The so-called “syphilitic phthisis” is usually tubercular. Cancer is often associated with tubercle. Patients dying of cancer frequently present tubercular deposits in their lungs.

Severe dyspepsia and anæmia may be either predisposing causes or early manifestations of phthisis. My experience tends to confirm the time-honoured observation that distaste for, or inability to assimilate, fatty foods is a frequent precursor of phthisis.

The above facts may sometimes assist us in doubtful cases. I pass on to consider the three lines of inquiry which are by far the most important in the early diagnosis of phthisis, viz. the consideration of—

C. The symptoms and mode of onset.

D. The sputum.

E. The physical signs.

C. The Symptoms and Mode of Onset.

I have already stated my opinion that phthisis may develop practically without symptoms. This is, however, very rare. As a matter of fact, our difficulty is usually the reverse of this—there are symptoms, and there are not signs.

* T. S. Clouston, M.D. : Mental Diseases, fifth edition, p. 507.

I bi d.

Can phthisis develop in the absence of cough? Probably we may answer this question in the affirmative, but such an event is certainly most rare. Just as a few isolated tubercles may exist in the lungs and yield no physical signs, so a similar condition may exist and yield no symptoms. Yet the cases are very few indeed where tuberculization of the lungs exists without exciting localized bronchial catarrh and, as a consequence, cough. In any suspicious case of debility or loss of flesh, if we can satisfy ourselves of the absence of cough from the beginning, the presumption is against phthisis. It must be borne in mind that some patients deny the existence of cough when it is really present, and that it is not uncommon for cough to be temporarily absent. I have very seldom discovered the existence of phthisis when an intelligent patient has assured me that cough had been absent from the outset, but, on the other hand, the disappearance of cough in favourable cases in the course of treatment is common. We must distinguish, then, between the entire absence of cough from the beginning and its disappearance under treatment. This latter fact has no diagnostic significance.

Absence of cough is, I take it, one of the most important negative points in the history. It is much more important than the absence of wasting, hæmoptysis, or night-sweating. This group of symptoms is most important positively, but only of minor importance negatively. Many cases of phthisis, it is almost unnecessary to remark, come on without hæmoptysis or night-sweating, and a smaller number come on without wasting.

Hæmoptysis is one of the most important positive points in the history. We shall never regret attaching great weight to it. The hæmorrhage may vary in

frequency and amount, but if the blood has undoubtedly come from the lungs, and there is no other obvious explanation of its appearance, it is a most important confirmation of the suspicion of phthisis.

Before allowing the fact of hæmoptysis to influence our judgment in a doubtful case, we should, of course, inquire whether any other explanation than phthisis is forthcoming—*e.g.* heart disease, anæmia, scurvy, hæmophilia, varicose veins of pharynx, Bright's disease, cirrhosis of the liver. In the absence of such conditions, hæmoptysis is in the immense majority of cases due to tubercular disease of the lungs. The "hæmorrhage from the throat," of which we hear so much, is usually mythical. The so-called "arthritic hæmoptysis" had better be viewed in most cases with considerable scepticism.*

In analyzing the history of attacks of hæmoptysis, one point in connection with phthisical cases has usually proved very helpful to me. The blood may be large or small in amount, may be arterial, venous, or capillary, but if the bleeding be at all considerable it does not cease suddenly, a free bleeding being most often succeeded by blood-spitting, lasting some hours, often some days. I have inquired into this point in a large number of cases, and I believe this rule may be trusted. It must be remembered, however, that it only applies to cases where the bleeding has been moderately free. Some patients give a history simply of a few casual spits of blood.

Pyrexia, impairment of appetite or digestive power, and wasting form a group of symptoms closely related to each other, and highly characteristic of incipient phthisis. If the patient has fever and disorder of digestion, he will certainly waste. If, on the other hand, as sometimes

* See Lecture XI., The Causes and Management of Hæmoptysis.

happens, he is free from fever and digestion is good, he will not waste. Upon the whole, however, wasting in some degree is one of the most constant symptoms, and we may say with substantial accuracy that in the diagnosis of early phthisis the weighing-machine plays a part hardly less important than the stethoscope and the thermometer. Wasting attended by fever—if not otherwise accounted for by some obvious condition, *e.g.* empyema, bone disease, sepsis—suggests the possibility of tuberculosis of the lungs; if to these symptoms cough be added, the suspicion becomes strong, and if to wasting, fever, and cough we add hæmoptysis, the suspicion becomes almost a certainty.

As regards the type of pyrexia in incipient phthisis, it almost always takes the form of a moderate rise of temperature in the afternoon. The practitioner will find it helpful to take the temperature occasionally after exercise. Rectal temperatures are more trustworthy than those taken in the arm-pit or the mouth.

Opinions differ as to whether well-marked and obtrusive dyspepsia is, in the absence of other symptoms, a frequent precursor of phthisis. Upon the whole, my opinion would be in the negative. If we reflect upon the history and course of the great army of dyspeptics, it would appear that the percentage of them who become tubercular is a small one—smaller than might have been expected on the theory that dyspepsia involves “lowered vitality,” and hence, probably, diminished resisting power to disease. As a broad rule, the dyspeptic remains dyspeptic, and does not become tubercular. The exceptions to this rule, though not numerous, are important. There is a type of dyspepsia where the gastric symptoms are not very marked, but where the patient becomes anæmic and wastes. A certain not inconsiderable proportion of such

cases ultimately become tubercular. The voluble dyspeptic who complains loudly of many miseries, but retains a fair degree of nutrition (the type, I may say without irreverence, immortalized in the letters of Thomas Carlyle), seems to me in no greater danger of tuberculosis than the healthy man. If, in connection with dyspepsia the possibility of tuberculosis be apprehended, the thermometer may give us important indications.

Pain in the chest, especially in the apical region, is common in incipient phthisis. It is often due to local pleurisy, often, I think, to nervous erethism. It may be accompanied by local tenderness.

Persistent frequency of the pulse in a young adolescent, not otherwise accounted for, should lead to inquiry for phthisis.

The combination of the following symptoms, languor, debility, and proneness to sweat on slight exertion, requires to be interpreted with reserve. With such conditions the suspicion of phthisis is apt to arise, but in the absence of cough, wasting, afternoon pyrexia, acceleration of the pulse, or a history of hæmoptysis, I do not think there is strong foundation for it. Obviously, under such circumstances the indications afforded by the stethoscope, thermometer, and weighing-machine should be scrutinized with special care. It is unfortunate to suggest the fear of tuberculosis to a patient who is suffering from "nervous debility." We may get assistance in such cases by asking the questions, Is this condition of debility recent or of long standing? Can any adequate cause be assigned for it—*e.g.* over-study, anxiety, prolonged nursing of sick relatives, disappointment? Incipient phthisis more often *appears* uncaused than does nervous debility.

Chlorosis is often viewed by anxious parents as the

harbinger of phthisis, usually without sufficient cause. Chlorotic girls do not become tubercular.

I have always a strong suspicion of commencing phthisis in the following type of case, of which I have seen many examples. The patient, generally an adolescent, protests that he is practically quite well, and submits with reluctance to examination. He at first denies all symptoms, but after a little conversation admits that he has a little cough in the mornings, that on one or more occasions he has seen a trace of blood in the sputum (but he is sure "it came from his teeth," or "he had a bleeding from the nose the day before"), that he has lost weight lately (but this is due to "having cycled so much," or to late hours of work, or so forth), he perspires rather freely at night (but this "is due to the hot weather"). The chest is found to be rather flat and inexpandible, the temperature is slightly raised in the afternoon, the patient is thin, he has a bright eye, a slight flush over the malar bones, his eyelashes are long and silky, his hands are rather hot, his intelligence is alert and above the average. We all know the type. In such cases the physician does not need to be a wizard to detect the projected shadow of tuberculosis.

D. The Examination of the Sputum.

It is hardly necessary to insist upon the primary importance of an examination of the sputum in all cases of suspected incipient phthisis. No doubt in a large majority of cases the symptoms and physical signs are in themselves decisive, but the detection of tubercle bacilli will sometimes at once clear up cases where symptoms and signs are ambiguous, and in a certain small minority of cases bacilli may be found where the confirmation of tuberculosis from other sides is practically *nil*.

I do not propose to enter into this subject in any

detail, but a few rules may be laid down regarding it from the clinical side.

1. Bacilli may be present in the sputum from the very earliest stage. This is, however, quite exceptional.
2. Failure to find bacilli must not be taken as evidence against tuberculosis, if the grounds for that diagnosis are otherwise strong.
3. Bacilli are occasionally present in the blood of early hæmoptysis—more often not.
4. Phthisis may advance a considerable way and be attended by free expectoration, and yet bacilli may not be found.

E Physical Signs.

In connection with incipient phthisis, we shall do well to regard the localization of the signs as more important than their character.

Signs, whatever their precise character, which tend to be confined to one apex, or if present on the two sides are much more pronounced on one, or which involve chiefly the upper portion of the lower lobe or lobes, or which begin in a median or basic position, but creep upwards towards the apex, are almost certainly due to tuberculization.

I have already expressed the view that a certain group of signs—viz. flattening, some loss of percussion resonance, and a few crepitant (consonating) râles at one apex—indicate, not an early, but a somewhat advanced stage of the disease. The flattening points to retraction of the lung, the dulness and consonating râles to a certain degree of consolidation of the lung. These conditions do not develop until tuberculization has made considerable progress.

In certain cases which the history would suggest as probably early I have met with quite a different group of

signs, viz. unimpaired movement, slightly increased resonance on percussion, and numerous fine bubbling, or faintly consonating, râles, such signs being often confined to one infraclavicular region. These signs may easily mislead. They probably point to a recent but somewhat active tuberculization of one upper lobe.

The earliest physical signs of incipient phthisis are not always the same. Tubercular invasion of any organ is "asystemic." It does not respect anatomical boundaries or physiological frontiers. Bacilli reach the lung by two channels—

(a) By way of the blood or lymph stream.

(b) By way of the air passages.

In the former case the alveoli are first affected, in the latter, the finer bronchi and bronchioles. The earliest effects of tubercular invasion of the lungs would, therefore, seem to be a catarrh limited to certain alveoli or bronchioles. With this conception in our mind, it is evident that flattening, dulness, and consonating râles cannot be the signs of commencing tubercular infiltration of the lungs. These signs presuppose changes in the lung which require time. We should expect, *a priori*, that the earliest signs of commencing phthisis would be—

(a) Certain alterations in the breath sounds.

(b) Fine bubbling (soon to become crepitant) râles.

Clinical experience will be found to confirm this expectation if we rid our minds of the fallacious picture of incipient phthisis characterized by flattening, dulness, and localized crepitant râle.

(a) *The breath sounds.*

Much attention should be devoted to the state of the breath sounds in a case suggestive of incipient phthisis. We must be fully alive to the possible limits of physio-

logical variation. The following rules will be found helpful:—

The alterations are local—nearly always apical.

The breathing may be harsh, weak, or cog-wheel.

The expiration is usually somewhat prolonged.

Coughing brings out the breath sounds with increased clearness in those cases in which the breathing is weak.

It has been doubted whether harsh breathing or weak breathing is the earlier condition. After much attention to the subject, I am inclined to think that there is no fixed rule. Both are frequent, both are often early; it is doubtful if either is the invariable antecedent or sequent. It cannot, I think, be truly said that harsh breathing always gives way to weak breathing, or *vice versa*. This point is worthy of the most careful consideration, and if it could be proved that either harsh breathing or weak breathing invariably comes first, and is succeeded by the other, the labour of diagnosis would be much lightened.

Cog-wheel breathing occurring alone is not a trustworthy sign, but it is certainly common in incipient phthisis, and cannot be dismissed as a sign of absolutely no significance.*

(b) *Râles*.

A fine bubbling, or obscurely crepitating, *râle*, limited to a small area of one apical region, often audible only after coughing, is usually the earliest sign. When we hear a limited, rather coarse, and definitely consonating *râle*, the case is no longer incipient. This *râle* may vary from day to day. It may come and go, and this variability may raise unfounded doubts regarding the

* I cannot, therefore, agree with the statement of Gee that cog-wheel breathing "is a sign of no practical importance."—Auscultation and Percussion, third edition, p. 127.

correctness of the diagnosis. It is important to insist that the detection of this form of *râle* is not necessary to the diagnosis.

I have stated my opinion that when well-marked dulness or percussion is found at one apex, the case is no longer incipient. What are we to say of the slighter departures from normal percussion resonance? I have obtained much assistance in the diagnosis of incipient phthisis by attention to this point. In many cases, on first percussing the infraclavicular regions on the two sides, our ear does not detect any obvious dulness, but on close attention to the sounds we are conscious of a difference on the two sides. On one side or the other the note seems, not dull, but slightly flat in tone and of raised pitch. This is usually the side of commencing disease. Hence I cannot subscribe to Strümpell's statement, "He who lays too great stress on the uncertain results of percussion will often make a false diagnosis."* The results of percussion, when the changes are slightly marked, are difficult to appraise, but they are most important.

Lowering of the upper limit of pulmonary resonance at one apex is a sign of importance. This point is not easy to establish, but should be borne in mind.

The following passage from the well-known and excellent work of Fowler and Godlee calls for some observations: "The diagnosis may be attended with difficulty when the symptoms suggest tubercular disease, but the physical signs are of doubtful import, and there is no expectoration. The cases in which the above conditions are fulfilled are very often delicate-looking young women suffering from anæmia. In such cases, and in all others included in this category, it is a golden rule never to make a diagnosis from tuberculosis from doubtful

* A Text-book of Medicine, English edition, p. 252.

physical signs, and particularly not from a single sign. The examination of the chest must be systematic; the evidence obtained on inspection and palpation must be considered with, and must agree with, that derived from percussion and auscultation. When the signs separately considered are of doubtful import, but are yet in harmony, and all point in one direction, a positive opinion may be given. But the observer *must beware of relying upon auscultation, with its many fallacies*,* and a definite opinion should rarely be given in doubtful cases, unless the record of the morning and evening temperature for at least a week is available."† Much of the above is quite sound—some of it goes without saying—but if we are always to wait until all the above conditions are fulfilled, we shall seldom make a diagnosis of incipient phthisis. Feeling strongly as I do the disastrous results of a procrastinating policy in these cases, I am unable to subscribe to rules which would make an early and prompt diagnosis often impossible. We need not expect that in incipient cases the signs obtainable by inspection, palpation, percussion, and auscultation will present a consistent and congruent whole. To distrust auscultation is not a safe rule. To withhold an opinion in the absence of afternoon pyrexia is not always necessary. Tuberculization of the lung may proceed a considerable distance without pyrexia. We must act here upon the rule which has such a wide application in practical medicine—viz. attach much weight to the presence of certain signs, little weight to their absence. In a doubtful case, the presence of afternoon pyrexia is confirmatory evidence of the highest value. Its absence is to be noted, but must not be allowed to prejudice the diagnosis.

* The italics are mine.

† Fowler and Godlee: The Diseases of the Lungs, p. 378.

It will be remarked that while Strümpell cautions us against over-confidence in the results of percussion, Fowler advises us to distrust auscultation.

In cases such as these, which are often of extreme difficulty, but where the necessity for a prompt diagnosis is urgent, these general axioms do not really help us. No doubt, either percussion or auscultation may mislead, but there is nothing gained by discrediting these methods in advance. It is certainly true that where the signs of incipient phthisis are slight and vague, we should attach weight to the corroboration of one class of sign with another, but this is a self-evident principle.

Leube * is of opinion that an apical catarrh is almost invariably tubercular, and that one of the most constant signs of such catarrh is a slight degree of dulness in the supraclavicular region. He believes, however, that a very slight degree of difference of percussion-note at the apex is found in conditions other than tuberculosis—*e.g.* emphysema. He thinks the diminution in the height of percussion resonance above the clavicle significant. He proceeds as follows: "Noch wichtiger für die Sicherheit der Diagnose ist, wenn *einer Abweichung des Percussionsschalls auch auscultatorische Abweichungen vom Normalen entsprechen*. Es genügen hier schon leichteste Veränderungen des Athmungsgeräusches: saccadirtes Athmen, verlängertes Exspirium, schwaches oder verschärftes oder rauhes Vesiculärathmen, unbestimmtes Athmungsgeräusch. Hat das letztere vollends einen bronchialen Charakter oder finden sich Rasselgeräusche, auch wenn dieselben ganz vereinzelt und nicht klingend sind, so gewinnt damit die leichteste percussorische Dämpfung der Lungenspitze hohe diagnostische Bedeutung."

Of cases of apical catarrh, where definite proof of

* William v. Leube: *Specielle Diagnose der Innern Kraukheiten*, p. 149.

tuberculosis was wanting, I have seen several examples, but in no single case do I feel certain that it was non-tubercular. In one instance in my practice a well-defined apical catarrh in a young lad was followed by apparently complete recovery and disappearance of physical signs, but eight or nine years subsequently he died of phthisis.

In connection with such cases croupous pneumonia affecting one apex will not give rise to any difficulty. Broncho-pneumonia confined to one apex is almost certainly tubercular.

No rule is more borne in upon us by an extended experience than that we should regard with extreme suspicion localized signs, which are limited to, or tend to concentrate themselves upon, one apex, either of the upper or the lower lobe.

The increased conduction of the heart-sounds to one apex has been thought by some observers to be a point of diagnostic importance in connection with incipient phthisis. I have not found this sign of any definite value in practice.

Increase of tactile fremitus, on the other hand, in one apical region is always deserving of consideration. We must carefully remember that in health the fremitus is often much more marked on the right side than upon the left. This sign will assist us more in investigating a suspicious left apex than a suspicious right apex.

Variation in the physical signs in incipient phthisis.

When we realize that the physical signs of incipient phthisis are due to a limited apical catarrh, there is no difficulty in comprehending that these signs may present variations from time to time. The catarrh is due, in the first place, to the irritation caused by bacilli, but it is to

some degree affected by such conditions as weather, state of the general health, amount of co-existing general bronchitis, and perhaps other factors. Variations in the physical signs does not, then, in every case imply variations in the extent or the activity of tuberculization.

Such variations may be due to—

1. More or less secretion in the bronchial tubes, due to weather, occupation, state of the general health.
2. Compensatory emphysema of the sound lung. Dulness may become hyper-resonance, the position of the heart may undergo some change, the state of the breathing and of the adventitious sounds may vary.
3. Actual changes in the diseased areas.

Slight variations in physical signs in incipient phthisis do not either on the one hand discredit the diagnosis, nor upon the other necessarily throw any light upon prognosis.

Radioscopy in the diagnosis of incipient phthisis.

It is claimed on behalf of radioscopy :—

- (1) That unilateral limitation of diaphragmatic movement as seen by means of the fluoroscope is often the earliest indication of commencing pulmonary tuberculosis.
- (2) That by the aid of the Röntgen rays pulmonary tuberculosis can be diagnosed at an earlier stage than by any other means at our disposal.*

Dr. A. Bécclère, who has given the subject much attention, writes as follows :—

* On the Use of the Röntgen Rays in the Diagnosis of Pulmonary Disease, by J. F. Halls Dally, M.A., M.D., *Lancet*, June 27, 1903. See also Dr. Walsham's articles, and those of Bécclère, Costa, Espino y Capo, Bonnet Leon.

"The Röntgen rays are particularly valuable in cases where tuberculosis is only suspected in the very early stage. Examination by the radioscope and radiograph supersedes all other methods, and shows a diminution in the clearness of the lung at one of the apices, often accompanied by a diminished descent of the corresponding half of the diaphragm; and for a certain time this last symptom may be the only one observed. More often this new method confirms the record made on auscultation, and shows that attending the slight and dubious modifications of the respiratory murmur there is positive condensation of the pulmonary tissue. However, in other cases auscultation and percussion forestall Röntgen rays in proving change in the lung." As regards the different methods of carrying out examination by the Röntgen rays, the same writer says: "These different methods, radioscopic examination, simple radiography, cinemato-radiography, and stereoscopic radiography, are of mutual assistance to one another in diagnosing thoracic affections, but to the medical practitioner they are not all of equal importance. Of the above methods, radioscopic examination is the simplest, easiest, quickest, and least expensive. But that is not all; it surpasses all other methods in the amount and importance of the information it gives in a short time. Hence it comes first, and more often than not by its use in diagnosis it is possible to dispense with the others." *

Dr. Bonnet Leon says: "M'appuyant sur de nombreuses observations, j'estime que, en dehors de toute altération visible du poumon, une anomalie respiratoire quelconque dans le jeu du diaphragme principalement, doit, lorsqu'elle est constatée à l'écran, toujours éveiller l'idée de tuberculisation." †

* Les rayons de Röntgen et le diagnostic de la tuberculose. Paris, 1899.

† Transactions of the British Congress on Tuberculosis, vol. iii. p. 287.

It would be premature to attempt to pronounce judgment upon these claims on behalf of the use of radioscopy in pulmonary disease. The method is still in its infancy. Much depends upon the completeness and excellence of the technique. We must await the verdict of a wider experience.

LECTURE VII.

CONDITIONS WHICH SIMULATE PULMONARY PHTHISIS.

SUMMARY.

Three groups of cases which simulate pulmonary phthisis.

- A. A group which may closely simulate pulmonary phthisis, but which, owing to their infrequency, give rise to little difficulty in practice, viz.—

Actinomyces, malignant disease, syphilis.

- B. A group which may closely simulate pulmonary phthisis, but in which, owing to well-marked differences in history, symptoms, and physical signs, the differential diagnosis is, as a rule, easy, viz.—

Pulmonary collapse, bronchiectasis, pulmonary embolism.

- C. A group which may closely simulate pulmonary phthisis, and in which the differential diagnosis often presents great difficulty, viz.—

Broncho-pneumonia, pleurisy, bronchitis, asthma, anæmia, neurotic dyspepsia.

Rules for differential diagnosis, canons and cautions.

THE conditions which may simulate pulmonary phthisis are many.

It will be of some practical service if we classify them into groups, as in the foregoing summary.

We have a few rare conditions, where the simulation of phthisis may be close, but which practically give rise to little difficulty, viz. actinomyces, malignant disease, syphilis. We have some conditions which are, upon the

whole, readily differentiated, viz. collapse, bronchiectasis, embolism. And, finally, we have some common conditions which sometimes cause serious difficulties in diagnosis, viz. certain cases of broncho-pneumonia, pleurisy, bronchitis, asthma, anæmia, and various neuroses.

A. The first group includes the following cases :—

Actinomyces, cancer, syphilis.

Actinomyces.

The lungs are affected in about 13 per cent. of cases of actinomyces. In only a very small proportion of cases is the pulmonary affection primary. The course of these cases may present a somewhat close simulation of that of phthisis. While the diagnosis of actinomyces can be certainly made only by detecting the presence of the ray fungus either in the sputum or in the discharge from an abscess, attention to the following considerations will usually serve to prevent confusion between this rare disease and phthisis.

1. The patient's occupation. About 75 per cent. of cases of actinomyces occur in persons who in the course of their occupation have to deal with cereals, viz. labourers, millers, farmers, and grooms.

The source of infection is usually one of the grasses, especially of the genus *Hordeum*.

2. The presence of actinomycotic lesions in other organs, *e.g.* jaw, tongue, mouth, intestines, liver, skin.
3. The detection of the ray fungus in the sputum or the pus from abscesses.
4. The involvement of the chest-wall. This is frequent in actinomycosis.

5. The localization of the physical signs in the lungs.

In actinomycosis the physical signs are usually basic. They do not tend to invade the apices, nor do they obey the laws of "the march of the lesion," which are so characteristic of phthisis.

6. The favourable influence of iodide of potash upon actinomycosis.

It must be borne in mind that actinomycosis and pulmonary tuberculosis are not uncommonly found in the same patient.

Malignant Disease of the Lungs.

Malignant disease of the lungs is a very rare condition. I have met with three examples.* Practically it is always secondary to malignant deposit elsewhere in the body, *e.g.* mamma, uterus, bones, glands.

The simulation of pulmonary phthisis by malignant disease of the lungs may be very close. In both conditions we may have cough, hæmoptysis, pyrexia, night-sweating, and wasting. The physical signs of the two conditions, though usually more or less contrasted, cannot always be distinguished with certainty. The history, though usually very helpful, is not always decisive.

We may hope to distinguish malignant disease of the lungs from phthisis by attention to the following points:—

- I. There is a history of pre-existing malignant disease in the mamma, uterus, bones, glands, or other organs.

* For a fuller discussion of this subject, see Lecture XII., "Some of the Rarer Forms of Pulmonary Disease."

In the present lecture only the points of importance with regard to differential diagnosis are considered.

2. Pain is a prominent feature. It is usually severe, persistent, and localized.
3. Pressure-signs tend to develop, but may be late in appearance.
4. The physical signs in the lungs vary much. They may simulate bronchitis, pleurisy, or phthisis. If they simulate phthisis, *i.e.* if they consist of areas of dulness, bronchial breathing, and crepitation, it will be found that these signs do not follow the laws of the localization of tuberculosis, either as regards the parts primarily affected or "the march of the lesion." In malignant disease the signs are most often in the substance of the lung, median or median-basis in site.

Pleural effusion is common in both malignant disease and phthisis, and in both cases the effusion may be bloody.

5. The sputum in malignant disease may be mucoid, muco-purulent, or sanguineous. The "red-currant jelly" expectoration is said to be somewhat characteristic; tubercle bacilli are absent.
6. Cachexia is rapid, marked, and progressive. A few months commonly serve to bring the patient to a condition of profound dyscrasia.
7. Treatment is usually quite unavailing, and the progress of the case does not exhibit those alternate amendments and relapses which are common in phthisis.

Pulmonary Syphilis.

Pulmonary syphilis is an extremely rare condition. Only a few undoubted cases are on record.

The diagnosis of pulmonary syphilis from phthisis will be assisted by attention to the following points:—

1. The history of syphilitic infection.
2. The co-existence of syphilitic lesions of larynx, bones, liver, spleen, or testis.
3. The localization of the lesions, which do not present the well-known peculiarities of tuberculosis, either as regards the seat or seats of the primary deposit or "the march of the lesion."
4. The nature of the lesions. Tubercular lesions tend to excavate, syphilitic lesions do not.
5. The presence in an important proportion of syphilitic cases of stenosis of the trachea or bronchi.
6. The absence of tubercle bacilli from the sputum, which is often profuse and foetid.
7. The general progress of the case, the constitutional condition of the patient, and the results of treatment.

Tuberculosis is a not uncommon development in the course of chronic syphilis.

- B. The second group includes the following cases :—
Pulmonary collapse, bronchiectasis, pulmonary embolism.

Pulmonary Collapse.

This condition may usually be diagnosed from phthisis by attention to the following points :—

1. The history of the case. In collapse there is usually a history of—
 - (a) Such conditions as acute bronchitis, broncho-pneumonia, measles with pulmonary complications, whooping-cough ; or
 - (b) Pressure on the lung or bronchi by tumour or aneurism ; or
 - (c) Foreign body in the larynx or bronchi.

2. The nature of the symptoms. Dyspnœa is prominent in collapse, not usually prominent in phthisis. Hæmoptysis is rare in collapse. Pyrexia may be absent in collapse, or if present, will be conditioned by the antecedent condition, and will not present the characteristic curve of phthisis.
3. The nature of the physical signs. In collapse we usually find retraction of the side, weak or faulty bronchial breathing, and diminution of local fremitus and vocal resonance.
4. The localization of the physical signs. In collapse the signs are usually basic or median-basic, very rarely apical.

Bronchiectasis.

In this condition there will probably be a history of prolonged bronchitis, of the discharge of large quantities of putrid sputum, without bacilli (hæmoptysis is not uncommon), the constitutional condition is often fair, there is no characteristic pyrexia, the physical signs are usually basic in seat, and consist of cavernous or diffuse blowing breathing, and amphoric phenomena. The character of the sputum is the most important diagnostic point. Bronchiectasis is a common feature of advanced phthisis. In such cases it is usually apical in seat. The history of the case, and the examination of the sputum will usually clear up these cases.

Pulmonary Embolism.

In certain rare cases a pulmonary embolus may give rise to localized crepitant râles, which may suggest phthisis, but the presence of mitral disease, the course and history of the case, and the examination of the sputum will usually make the differential diagnosis easy.

C. The third group includes the following cases :—

Broncho-pneumonia, pleurisy, bronchitis, asthma, certain neuroses.

Broncho-pneumonia.

The distinction of broncho-pneumonia from phthisis is often difficult, and sometimes impossible. Nor is this fact surprising. Phthisis is a form of broncho-pneumonia, but of special character and specific origin.

It is well to recognize that the physical signs of broncho-pneumonia may be identical with those of phthisis. In many cases, also, the symptoms of the two conditions are indistinguishable.

Assistance may be obtained from attention to the following points :—

1. The history in broncho-pneumonia will probably cover a period of days or weeks (not months or years, as in phthisis).
2. The co-existence of some of the frequent causes of broncho-pneumonia — *e.g.* measles, whooping-cough, rickets. It must be borne in mind, however, that some of these conditions are not rarely associated with phthisis.
3. The symptoms in broncho-pneumonia are often more acute and obtrusive than in phthisis.
4. The curve of the temperature, though often not characteristic, may assist the diagnosis.
5. There are no tubercle bacilli in the sputum in broncho-pneumonia.
6. The physical signs in broncho-pneumonia are usually bilateral from the outset.

The question of the differentiation of broncho-pneumonia and phthisis usually arises in the following form :—

A child has passed through an attack of measles,

whooping-cough, typhoid fever, or other disease. Pulmonary complications have been present throughout. The convalescence is lingering, the constitutional condition indifferent or bad, and certain physical signs persist in the lungs. The question arises, Is the case simply one of delayed convalescence, or has tuberculosis set in?

The following points will assist the diagnosis:—

1. The sputum. The presence of tubercle bacilli is, of course, decisive of tuberculosis; their absence upon repeated examination would be strongly in favour of broncho-pneumonia.
2. The physical signs must be very cautiously appraised. Often no secure conclusion can be drawn from them. In broncho-pneumonia the signs will be almost without exception bilateral from the outset, and will show a preference for the basic or median-basic situations. Any tendency for the physical signs to limit themselves to one in both apices has, in my experience, almost invariably coincided with the development of tuberculosis. This rule cannot, however, be reversed with safety. I have seen a small number of cases where the physical signs began at the base and gradually spread upwards, and in which the subsequent development of the cases left no doubt as to their tubercular character. Such cases are, however, rare.

Dulness, bronchial breathing, and crepitant râles are equally characteristic of broncho-pneumonia and phthisis. Signs of commencing excavation, not always easy of recognition, would point to phthisis.

3. The symptoms require to be interpreted cautiously. Hectic fever, wasting, and night-sweating may

all be well marked in non-tubercular cases. Hæmoptysis, if present, would go far to determine the diagnosis, but this symptom is often absent in the commencing phthisis of young subjects.

A point upon which I have sometimes relied with success is the following : The tubercular patient is *less obviously ill* than the patient with simple broncho-pneumonia. His symptoms and his physical signs are less accordant than in the latter condition.

This rule requires to be applied with reserve, but it has a certain value.

When all has been said, the differentiation of broncho-pneumonia from phthisis is sometimes one of the most difficult tasks of the physician. Osler does not overstate the difficulty when he says, "It is well to emphasize the fact that there are many cases of broncho-pneumonia in children which time alone enables us to distinguish from tuberculosis." *

Pleurisy.

Bearing in mind that a large proportion of pleurisies are tubercular in origin and that a certain degree of pleurisy always accompanies phthisis, it is not surprising that great difficulty may be encountered in differentiating the two conditions. Sometimes we may be seeking for a means of distinguishing two conditions which are fundamentally one.

Nevertheless, the question of the differentiation of pleurisy from phthisis is one of great practical importance.

It commonly arises in one or other of the following forms :—

* Practice of Medicine, fourth edition, p. 646.

- (a) During the course of an acute pleurisy crepitations are heard at the apex in the affected side, and the question of the possibility of phthisis arises. It should be a cardinal rule to give no positive opinion regarding these cases during the continuance of the effusion. The signs in question are often due to adhesions of the lung at the apex.
- (b) A patient has passed through an attack of acute pleurisy, convalescence is tedious, expansion of the lung is imperfect, perhaps some residual effusion persists. Are we dealing with the residue of a so-called simple pleurisy, or has tuberculosis set in?

This question may not always admit of a definite answer. The sputum must, of course, be repeatedly examined. A persistently hectic temperature is suspicious of tubercle, but the absence of fever may be consistent with either diagnosis. If fluid be present it may be examined both microscopically and, if possible, by inoculation. The fluid may be serous, bloody, or purulent. A serous effusion throws no light on the differential diagnosis. A bloody effusion lends weight to the suspicion of tubercle. A purulent effusion is usually non-tubercular. The bacillus of tubercle is very rarely found in pleural effusions. The results of inoculation experiments may be decisive.

The local physical signs may present nothing pathognomonic, but any tendency of the signs to extend towards the apex or any development of fresh signs at one or both apices is strong confirmation of the suspicion of tubercle.

Where a positive opinion is impossible, it is better to lean towards the more hopeful view and to treat these cases simply as chronic pleurisies. The treatment thus adopted,

viz. hygienic measures, pulmonary gymnastics, upland or mountain air, will, in most cases, be useful, even if tuberculosis should ultimately prove to be present. If the case be really tubercular, it will often be found that moderate exercise raises the afternoon temperature by a degree or two.

Bronchitis.

The differentiation of bronchitis from phthisis is usually a simple matter. In bronchitis the physical signs are bilateral, usually equally marked on the two sides, more pronounced at the base than the apex ; the tendency is toward over-expansion and hyper-resonance of the chest ; the râles, in the absence of complications, are bubbling and not crepitant.

In all these points phthisis presents a contrast. The signs are at first unilateral, never equally marked on the two sides, more pronounced at the apex than at the base ; the tendency is towards retraction and deficient resonance of the chest ; the râles are crepitant in character.

The departures from the above rules are few.

It must be admitted, however—

1. That in certain extremely rare cases bronchitis is for a time unilateral.*
2. That during convalescence from bronchitis the signs sometimes become localized before clearing up. If in such cases the constitutional condition be unsatisfactory, the suspicion of phthisis is likely to arise.
3. That phthisis may set in with "bronchitic" physical signs.

One is reluctant to admit the validity of these axioms, but there can be no doubt of their accuracy.

* S. Gee : Auscultation and Percussion, third edition, p. 202.

In such cases we may obtain assistance from attention to the following points :—

1. The presence or absence of tubercle bacilli.
2. The expansion of the chest—normal or increased in bronchitis, diminished in phthisis.
3. The state of the vocal fremitus—normal in bronchitis, probably increased in phthisis.
4. The percussion sound—hyper-resonant in bronchitis, deficient in resonance in phthisis.
5. The nature of the râles—bubbling in bronchitis, crepitant in phthisis.
6. The temperature, weight, presence or absence of hæmoptysis, night-sweating.

A hectic temperature, marked wasting, and night-sweating may all occur in the bronchitis of debilitated subjects. Hæmoptysis is strong presumptive proof of phthisis.

In rare cases phthisis sets in with the physical signs of general bronchitis, and the diagnosis may remain in doubt until the examination of the sputum or the occurrence of hæmoptysis clears up the case. An instance of this kind has recently occurred in my practice.

Asthma.

The association of asthma and tuberculosis is a rare one. The differentiation of the two conditions is usually obvious. Nevertheless, there are exceptions to these rules, and these exceptional cases are, from their rarity, apt to mislead.

The following type of case is occasionally met with in practice :—

A limited and retrocedent tubercular lesion of one apex is attended by fibroid changes and bronchiectasis. Attacks of dyspnœa occur, and the case is mistaken for one of asthma.

In such cases the differential diagnosis is usually easy. The history of the two types of case is quite different. The "asthma" in the tubercular case is rarely typical. There is more or less debility and emaciation. The temperature may assist. Tubercle bacilli may be found in the sputum, but are often absent. Localized signs at one or both apices, however slight, can be recognized.

The bronchiectatic changes in the neighbourhood of the apical lesion may produce misleading physical signs.

It should be a rule to examine the sputum for tubercle bacilli in cases of supposed asthma, and to exercise special care when the patient is young and when debility or emaciation is present.

Anæmia, Dyspepsia, various Neuroses.

These conditions, which may be conveniently considered together, often simulate incipient phthisis, and the difficulties of a differential diagnosis may be great. In one type of case anæmia and dyspepsia, in another nervous phenomena, are the prominent features. In many cases the two groups of symptoms are conjoined.

A typical case may be sketched as follows:—

A young girl (18 to 25 years of age) has been getting out of health for weeks or months. She has lost flesh, and is becoming anæmic. Dyspepsia—often under the form of pronounced anorexia—is marked. She has a short cough, and occasionally there has been a little blood in the expectoration. The temperature is normal, there is no night-sweating, and no physical signs in the chest.

Such a case may simulate phthisis, and if there should be tuberculosis in the family the fears of the patient and her friends may be acute.

The above type of case may be—

1. Genuine incipient phthisis.
2. Hysteria, with anæmia and debility.
3. Nervous mimicry (*i.e.* phthiseophobia).

It is important to observe that the simulation of pseudo-phthisis (if I may employ so questionable a term) to the genuine disease may be very close. Anæmia, wasting, debility, and dyspepsia may be marked in both conditions. Cough and hæmoptysis may also be present in both—a fact not always borne in mind. Yet on carefully analyzing the symptoms points of difference will usually appear.

The cough of incipient phthisis is somewhat persistent, but not usually incessant and obtrusive; it is dry and unsatisfying, and accompanied by slight mucoid or pearly expectoration. The cough of pseudo-phthisis is incessant and obtrusive, hard and clanging in character, without expectoration or with expectoration mainly composed of saliva.

Hæmoptysis is often the chief source of error, and it is important to bear in mind that hysterical girls may develop hæmoptysis (apart from organic disease of heart, lungs, or other organs) in two ways:—

1. By sucking their gums until blood comes.
2. By a genuine bleeding from the lungs due to vaso-motor disturbance.

The former of these modes is common, the latter is extremely rare.

The spurious cases do not develop pyrexia, or night-sweating, and, of course, there are no physical signs in the chest and no tubercle bacilli in the sputum.

In pseudo-phthisis the patient complains much of her symptoms, in genuine phthisis often she complains little. The former draws our attention to her symptoms, the latter

glosses them over. The former solicits physical examination, the latter shuns it.

If the blood of an attack of hæmoptysis can be inspected, a diagnosis may sometimes be made. In some cases the watery state of the blood and its manifest admixture with saliva may give us the necessary hint. Hysterical young women are fond of bringing handkerchiefs with a few dubious stains to the doctor. The genuine consumptive does not often do so. The former invites a serious diagnosis; the latter dreads it.

We must be specially on our guard in connection with cases where the patient is manifestly neurotic or hypochondriacal, but where actual disease may, nevertheless, be present. A neurotic girl may become tubercular, like any one else, and the presence of an obvious neurosis must not blind us to the possibility of organic mischief. Yet, in general, we have to deal either with commencing phthisis or mere neurosis, not with both.

In such cases as Anorexia Nervosa the extreme degree of emaciation present may strongly suggest tubercle, but if we are familiar with such conditions no special difficulty need arise in this connection.

In cases of doubt as regards the differential diagnosis of pseudo-phthisis and genuine phthisis, it is not advisable to lay much stress either on family history or upon such points as "tubercular aspect," "flat chest," or the like, for it is precisely where the family history or the patient's physique presents special ground for uneasiness that the spurious cases are most apt to arise. Indeed, we must exercise the greatest caution when we can perceive some obvious ground for the patient's agitations.

In these difficult cases it is most important not to be betrayed into a premature diagnosis. A second or a third examination and the regular use of the thermometer will

often be advisable. Under such circumstances, parents are often panic-stricken, and fully prepared for heroic measures. If, therefore, the physician without due warrant endorses their apprehensions, the patient may be forthwith haled off to a sanatorium, where the true nature of the case will soon become evident.

LECTURE VIII.

PROGNOSIS IN PULMONARY PHTHISIS.

SUMMARY.

Increased hopefulness of prognosis at the present day.

Influence upon prognosis of—

- (a) Age.
- (b) Sex.
- (c) Occupation and social position.
- (d) Family history.
- (e) Constitutional type of patient.
- (f) Mode of onset of the disease.
- (g) State of the lungs.
- (h) Extension of tuberculosis to other organs.
- (i) Symptoms.
- (j) Complications.
- (k) Response to treatment.

Unfavourable types of patient—

- (a) The erethic type.
- (b) The lymphatic type.

Favourable types of patient—

- (a) The “wiry” type.
- (b) The hæmorrhagic type.

Importance of habits.

Modes of onset in their relation to prognosis—

- (a) The insidious onset.
- (b) The acute onset.
- (c) The catarrhal onset.
- (d) The hæmorrhagic onset.
- (e) The consecutive onset.

Type cases, and the prognostic inferences to be drawn from them.

THE prognosis in pulmonary phthisis involves many difficult problems. The despair of past times has given place at the present day to a hopefulness which is not always

warranted. The pendulum would seem to have swung from one extreme to the other. It is well to remember that pulmonary tuberculosis is in all cases and under all circumstances a most grave condition, that the most promising cases may do badly, and that even when arrest seems complete, some casual "cold" or wayward hæmorrhage—some intercurrent and wholly incalculable malady—may disappoint apparently well-founded hopes, and relight the embers of quiescent fires.

Nevertheless, it is a great gain to recognize clearly that the disease is not necessarily incurable, and that even when treatment cannot cure, it may achieve much. Cure in the sense of complete *restitutio ad integrum* is, no doubt, a rare event, and practically confined to incipient and favourable cases. But cure, in the sense of relative cure, *i.e.* the arrest of the pulmonary mischief and the restoration of the patient to good general health and working power, for a longer or shorter period, is not by any means rare. And even when "relative cure" is not achieved, varying degrees of improvement, often marked and of considerable duration, are constantly obtained by treatment. Therefore the hopeful tone of modern therapeutics is, upon the whole, justified. We have learnt that pessimism and inaction are unjustifiable, that treatment will in a large proportion of cases repay its heavy price in time and money, and that the consumptive can make his appeal to us upon economic grounds, as well as upon those of natural humanity.

I shall first state the broad principles of prognosis about which there is more or less general agreement, and then enter more fully into those points which are to some extent *sub judice*.

Prognosis may be considered under the following heads:—

1. *Age*.—As a rule, the prognosis is worst in very young and very old patients. It is best, probably, in early adult life.
2. *Sex*.—According to most observers, the prognosis is more favourable in the male sex. It is probable that this is not an ultimate fact—*i.e.* a real sexual difference—but that it has relation to the habits, occupation, and mode of life of the two sexes. The influence of child-bearing requires consideration. That pregnancy, as has often been alleged, retards the progress of existing phthisis is doubtful. Prolonged lactation, undoubtedly, aggravates the disease. Phthisical women are, unfortunately, somewhat fertile.
3. *Family history*.—Many observers are of opinion that phthisis tends to arise at an earlier age and to run a more rapid course when the family predisposition to the disease is marked, but this is denied by others; and probably the influence of inheritance in these regards has been, upon the whole, overrated. It is obvious that the influence of inheritance may be difficult to disentangle from the influence of infection.
4. *Constitutional type of patient, previous health, occupation, social position, habits*.—These are, undoubtedly, factors of great importance. We shall resume their consideration presently.
5. *Mode of onset*.—An acute onset is always ominous, but does not necessarily portend an acute course. An insidious onset does not, in my opinion, throw much light on prognosis.
6. *State of the lungs*.—The larger the area of lung involved, the earlier the extension to the second

lung, the more the pathological process tends to softening and excavation—the worse the prognosis. Bronchiectasis is unfavourable.

Gangrene, which is rare, portends a rapidly fatal issue.

7. *Extension of the tubercular process to other organs.*—

Extension of the tuberculizing process to the larynx, intestines, kidneys, or brain, usually involves a definitely bad prognosis. Some laryngeal cases, however, do well.

Extension of disease to the glands has not much influence upon prognosis. Extension to the abdominal lymphatic glands is more serious than extension to the cervical, thoracic, or mediastinal glands.

8. *The nature of the symptoms.*—Marked pyrexia, wasting, marked loss of strength, rapid weak pulse, failure of appetite, diarrhœa—are unfavourable.

Certain forms of pyrexia are specially ominous—*e.g.* a very high afternoon temperature with a fall to several degrees below normal in the early morning. Hæmoptysis, unless profuse and frequently repeated, has not much weight in prognosis. The “bleeders” do at least as well as the “non-bleeders.” Of the various symptoms in phthisis, the most important with regard to prognosis is the state of the digestion.

9. *The complications.*—Amyloid degeneration of various organs—*e.g.* kidneys, intestines—is very unfavourable. Pneumothorax is usually fatal within a month, but in certain exceptional cases it seems to exercise a retarding influence upon the disease.

Albuminuria may be due to lardaceous disease of the kidneys, interstitial nephritis or tuberculosis of the kidneys, and is very unfavourable.

Ulceration of the bowels, peritonitis, diabetes, typhoid fever, puerperal septicæmia, usually involve a definitely bad prognosis.

Bronchitis, if moderate in degree, has not much influence upon prognosis; if severe, it is unfavourable, for two reasons: (*a*) it may point to a somewhat wide tuberculization of the lungs; and (*b*) it may embarrass treatment.

Pleurisy is not definitely unfavourable. In the opinion of some observers pleural effusion in the course of phthisis points to a probably chronic course of the disease.

Fistula in ano is not unfavourable.

10. *Response to treatment.*—As in many other diseases, one of the most essential facts in throwing light upon prognosis in phthisis is the response of the patient to treatment, which gives us some measure of what we vaguely call his “vital energy” or “resisting power.” Some patients at once begin to improve under more favourable conditions of life, and the adoption of a methodized hygiene. Others, not obviously more gravely affected, make little or no response. The difference between the two classes is, *quoad* prognosis, a very vital difference.

The above may serve as a brief outline of the factors which influence prognosis in phthisis.

Let us now consider the subject a little more closely, and enter into some of its more obscure and controversial aspects.

Age, sex, and family history are not matters of the

first importance from the prognostic point of view. I have known very few favourable cases of phthisis in patients under 15 years of age, but the influence of age does not follow any very definite rule. Senile phthisis does badly, as might be expected, and is not always chronic in its course. I am inclined to think that the most favourable age for phthisis is from 25 to 35. Patients between these ages sometimes respond well to treatment, while younger patients are apt to exhibit the disease in its more active forms, and older patients present special difficulties as regards the maintenance of nutrition.

Sex, not in itself a matter of great prognostic importance, may have influence indirectly, *e.g.* in its bearing upon treatment. Phthisis arising in a young girl of from 17 to 25 years of age involves more difficult problems from the therapeutic point of view than a case in the opposite sex within the same age limits. Travel, change of climate, the adoption of an out-of-door life and a healthy occupation, "roughing it" in a new country, are obviously more easily available for the male than for the female adolescent. Women do as well as men in sanatoria, nor, so far as I am aware, is there any difference in the results obtained in the two sexes at such resorts as Davos, or St. Moritz.

Family history requires a cautious appraisalment. Its influence has been overrated in the past, but it is in danger of being unjustly ignored in the present. It is probable that its importance depends mainly upon this consideration, *viz.* that families differ not only in their tendency to resist, or succumb to, tubercular invasion, but also in their capacity to withstand the disease when once established. A history that several members of a family have succumbed to phthisis either at an early age or after a brief illness

is very ominous. Much depends upon whether the activity of the disease in a certain family has been coincident with unfavourable hygienic conditions or not. The disease has been known to ravage a family when living under unhealthy conditions, but to cease entirely on removal to a more favourable environment. In other cases the disease has been known to continue to infect members of a family remaining at home, but has not pursued those who have left the family circle. It must be admitted, however, that exceptions to these rules occur. It is, however, true in not a few cases that "family predisposition" being interpreted in modern phrase might often read "house infection."

The patient's occupation, social position, economic resources, his power to obtain early and prompt medical advice and to adopt and persevere in appropriate treatment are factors of much prognostic importance. It is hardly necessary to labour this point, of which the relevance is obvious. It must be borne in mind that the prospects of treatment are better in cases where the patient's occupation is with just ground held to be directly responsible for his condition.

Early diagnosis and prompt and persevering treatment are obviously factors of great importance.

The constitutional type of the patient is a matter of much moment in connection with prognosis, but it is not easy to lay down rules which can be applied in practice. It would be quite erroneous to say that the typical thin, flat-chested consumptive always does badly; or that the well-developed and apparently robust patient usually does well. Some of the worst cases in my experience have belonged to the latter category. West says truly, "Gallop^{ing} phthisis may develop in a person apparently previously robust, and

on the other hand, in a weakly, delicate person phthisis may last for years." *

The following types seem to be relatively unfavourable:—

- (a) The nervous, irritable type—*eretische constitution*—the patient whose vaso-motor control is indifferent, who has hot hands and a moist skin, who sleeps badly, has a capricious appetite and an indifferent digestion, and whose capacity for fatigue is small.
- (b) The "lymphatic" type, *i.e.* the patient who does not at first waste or show much pyrexia, but who is obviously ill, whose symptoms of languor, debility, and malaise are out of proportion to the physical signs in the lungs, the pyrexia, or the wasting.

The following types seem relatively favourable:—

- (a) The "wiry" type, *i.e.* the patient who is thin but not debilitated, whose energy is but little impaired, whose appetite and digestive power are fair, whose pulse is good, and whose lungs seem prone to fibrosis rather than to softening and excavation.
- (b) The "hæmorrhagic" type, *i.e.* the patient who has either repeated small or occasional large bleedings, but whose general health remains fairly good, and whose lungs present few definite signs of disease.

There is nothing more difficult to gauge, more elusive of precise definition, than the "constitutional type" in any given cases of phthisis, yet we feel assured that it is a point of cardinal importance, that in fact the patient's personality weighs heavily in determining his prospects in the struggle with disease.

* S. West: Diseases of the Organs of Respiration, vol. ii. p. 535.

The mode of onset has great prognostic significance. The following modes of onset may be enumerated :—

- (a) The insidious onset, *i.e.* where the disease sets in very gradually, with ill-defined failure of general health, slight cough, loss of flesh and strength, dyspepsia, and perhaps some rise of afternoon temperature.
- (b) The acute onset, *i.e.* where the disease sets in rapidly with high fever, prostration, and speedy wasting.
- (c) The catarrhal onset, *i.e.* where bronchial catarrh is the leading feature of the initial stage.
- (d) The hæmorrhagic onset, *i.e.* where hæmoptysis is the earliest, or at least most outstanding symptom, of the initial stage.
- (e) The consecutive onset, *i.e.* where phthisis obviously follows upon measles, whooping-cough, broncho-pneumonia, or typhoid fever.

Speaking generally, I think it may be said with approximate truth that the following modes of onset are unfavourable, *viz.* the acute onset, the catarrhal onset, the consecutive onset, while the hæmorrhagic onset is somewhat favourable, and the insidious onset doubtful.

Upon these points, however, there is by no means general agreement.

Jaccoud is of opinion that pneumonic phthisis has a prognosis which "at first is more unfavourable than in any other form of the complaint, miliary granulosi alone excepted, while during the time which follows the acute outbreak it is the least serious of all the different varieties." *

Percy Kidd says, "An acute onset is commonly followed by progressive invasion of both lungs, and has the gravest

* S. Jaccoud : *Pulmonary Phthisis*, Lubbock's Translation, p. 43.

significance. An insidious, bronchitic, or hæmoptoic onset is more favourable." *

West says, "The most unsatisfactory cases are those in which the onset has been insidious, and where the patient has been losing health, flesh, and strength, without obvious cause." †

Wilson Fox says, "An acute pneumonic invasion with rapid consolidation of the apex rarely escapes softening and excavation." ‡

I incline to the view that an acute onset is, in general, decidedly unfavourable, though a small minority of these cases pursue a relatively slow and chronic course; that the insidious onset has no definite prognostic significance; that the bronchitic onset is decidedly unfavourable, and that the hæmoptoic onset is, with many important exceptions, somewhat favourable. The divergences of view quoted in the previous paragraph warn us, however, that hard-and-fast rules are not to be laid down in this connection.

The "consecutive onset" is unfavourable because in such cases we have usually to deal with a debilitated patient. No doubt, a good deal turns upon the precise nature of the pre-existing disease, *e.g.* phthisis supervening upon measles or typhoid fever is more unfavourable than phthisis supervening upon influenza or pleurisy. These rules are, however, of very limited value.

The rate of progress of the case has much prognostic import.

Evidence that the disease has lasted for a considerable period without serious impairment of the general health or the development of well-marked signs in the lungs is very

* Allbut's System of Medicine, vol. v. p. 226.

† S. West: Diseases of the Organs of Respiration, vol. ii. p. 535.

‡ Wilson Fox: Treatise on Diseases of the Lungs and Pleura, p. 842.

important. In such cases it will usually be reasonable to assume that the patient's resisting power is good, and that the tubercular invasion of the lung tends towards fibrosis, rather than towards softening and excavation. On the other hand, pronounced constitutional dyscrasia and wide involvement of the lungs occurring in the course of a brief space of time points to the opposite conditions, and is very unfavourable.

At the onset of a case of phthisis prognosis requires to be very guarded. Under such circumstances, we have as yet no gauge of the patient's staying-power. Of this we can form some estimate when we have watched the case for a time.

We cannot, however, pretend to define with any close accuracy the probable duration of a case of phthisis which is tending towards a fatal issue. We can tell what is the existing state of the lungs and of the general health, whether the probabilities point to a favourable or an unfavourable course, whether life is a matter of days or years, but the estimate of the duration of any given case is simply a guess, and a guess which is frequently wide of the mark. How often do we hear the story, "The doctor said I could not last one, three, or six months, and here I am after five, ten, or perhaps even twenty years." The chronic consumptive, the case where cure is manifestly impossible, journeys towards the inevitable goal over a chequered road. He has his ups and downs, his periods of comfort and quiescence, his periods of active disease and suffering; not rarely the grave seems to gape before him, but closes again; sometimes his flickering lamp of life is snuffed out all unforeseen. It is not well to attempt mathematical accuracy in our time forecast of such cases.

The state of the lungs is one of the most important points with regard to prognosis.

Extensive disease, early involvement of the second lung, the lighting up of fresh foci of tuberculization, the steady increase of the area of physical signs are of evil import. We cannot, however, reverse these rules. Ill-marked and limited physical signs are no guarantee of a favourable course of the disease. One of the worst types of phthisis is the case where constitutional symptoms—fever, wasting, debility—are out of proportion to the physical signs.

Further, marked and advanced signs, if of limited distribution, are not necessarily very unfavourable. Flattening, dulness, and bronchial breathing at one apex may be consistent with a limited lesion and more or less satisfactory arrest of the disease.

Signs of fibroid change are naturally much more hopeful than signs of increasing infiltration and softening. Hence, shrinkage of one apical region, with weak or faintly bronchial breathing, some dulness on percussion, and the absence of râles, constitutes a relatively favourable assemblage of physical signs, while the gradual extension of moist crepitant râles indicates the spread of the disease.

The extent of the physical signs requires to be interpreted in the light both of their nature and of their probable duration.

The presence of excavation is always more or less unfavourable. Yet a single cavity of moderate size at one apex with little involvement of the rest of the lungs constitutes a type of case where a prolonged course may not infrequently be anticipated. Cavities never close or become entirely obliterated, but they may dry up and remain quiescent for long periods. One of the best cases of arrest with complete restoration of health and practical abeyance of all symptoms for several years which have

come under my observation was that of a young lady who had a large cavity in the right infraclavicular region. Nevertheless, cavity cases, with very rare exceptions, ultimately do badly.

In a case where one apex is decidedly affected, too much weight *quoad* prognosis must not be assigned to evidence of slight extension to the apex of the other lung. Such an extension is, practically, a normal event, and is consistent either with a good or a bad prognosis. This is not the opinion of the laity, who are apt to be much discouraged if informed that "the other lung is affected."

Where physical signs and symptoms conflict as regards their prognostic indications, it is safer to trust to symptoms than to signs.

The nature of the symptoms [probably gives us the most trustworthy of all prognostic indications. Amongst the most unfavourable symptoms are the following :—

Severe and persistent pyrexia, especially if characterized by a high afternoon maximum and a very low morning minimum ; rapid and progressive wasting, especially if it ensue in spite of active alimentation ; failure of appetite, discomfort during digestion, diarrhoea ; rapid, weak pulse ; profuse night sweating ; profuse purulent sputum, containing many elastic fibres.

The following axioms may be laid down :—

The absence of pyrexia does not in itself warrant a hopeful outlook. In exceptional cases the disease progresses rapidly without fever. This is, however, extremely rare. A certain gain in weight is consistent with the steady progress of the pulmonary lesions. The hyper-alimentation of modern treatment not uncommonly produces the melancholy spectacle of a patient who has grown fat but not well. Nevertheless, the indications of the weighing-machine are among the most valuable of

prognostic aids. Marked dyspepsia is often due to dietetic errors. If it persists, in spite of a regulated dietary and careful treatment, the outlook is gloomy. Persistent frequency and feebleness of the pulse, in spite of rest, is very unfavourable. The number of tubercle bacilli in the sputum is no certain index of the activity or progress of the disease. Hæmoptysis, unless profuse and oft repeated, is not necessarily unfavourable. Dyspnœa is not very common. It may point to miliary tuberculosis, weak heart or profound debility, and hence be very unfavourable. Dropsy of the extremities generally marks the beginning of the end. *Spes phthisica* is sometimes marked in the worst cases.

The complications are highly important with a view to prognosis.

Amongst the gravest complications are the following :—

Meningitis, peritonitis, pneumothorax, ulceration of the bowels, amyloid degeneration of the kidneys, ulceration of the larynx, aphthæ of the mouth. Pleurisy, fistula in ano, various skin eruptions, are not unfavourable. Persistent diarrhœa is one of the commonest complications of phthisis. Its prognostic import is usually very grave, but varies with the cause. There are three main causes of diarrhœa in phthisis, viz. :—

1. Dyspepsia—especially fermentative changes in the stomach or bowels.
2. Amyloid degeneration of the bowels.
3. Ulceration of the bowels.

The differentiation of these causes is not usually difficult. The diarrhœa of amyloid degeneration of the bowels is a painless and intractable flux. The diarrhœa of intestinal ulceration is painful, there is tenderness in the abdomen, and perhaps blood in the stools. As

regards the prognostic inferences from these conditions: Dyspeptic diarrhœa may admit of cure, the diarrhœa of amyloid degeneration and of intestinal ulceration only of palliation.

Percy Kidd * thus sums up the prognostic problem: "The best results may be expected in cases presenting the following features: A pyrexia or a subfebrile temperature; weight stationary or increasing; signs of disease confined to one lung or to limited portions of both lungs (especially if associated with contraction); a quiet pulse and nervous system; a good digestion; absence of serious complications; a good family and personal history, and favourable hygienic surroundings."

I propose now to take a few type cases, and offer some observations on the prognosis.

CASE I.—A young adolescent (18 to 25) develops phthisis of the "insidious onset" type. There is cough, some loss of flesh, slight afternoon pyrexia, and some signs of commencing mischief at one apex.

Prognosis.—Doubtful, but not definitely bad.

The main aids to prognosis in such a case are the following:—

1. Whether the attack was predisposed to by hard work, a confined life, inattention to food, or came on in the absence of such predisposing causes.

The prognosis is better in the former case.

2. The state of the appetite and digestion.

Vigorous digestive power is highly favourable, and *vice versa*.

3. The state of circulation.

A pulse of good tension and moderate frequency is of favourable prognostic import, and *vice versa*.

* *Lóc. cit.*

4. The family history.
5. The patient's previous habits.
6. The prospects of securing suitable treatment extending over a sufficiently long period of time.
7. The prudence, self-control, and resolution of the patient.

CASE II.—A young adolescent (18 to 25) previously in apparent good health, is suddenly attacked by sharp pulmonary hæmorrhage depending on tuberculosis. The bleeding recurs once or twice, and then ceases. The general constitutional state is good; physical signs in the lungs are absent or obscure.

Prognosis.—Upon the whole, relatively favourable.

Death occasionally occurs at an early stage in such cases, but such an event is extremely rare.

If pyrexia follows the hæmoptysis and persists, it is probable that septic pneumonia is present, and the case passes into a less favourable category.

Pulse and digestive capacity are important.

These cases sometimes do well under various forms of sanatorium or climatic treatment.

They are suitable cases for the high altitudes.

CASE III.—Phthisis of a chronic type is known to have existed for six or twelve months or longer in a patient of from 20 to 30 years of age. There are signs of a well-marked but quiescent lesion at one apex. The general constitutional state is fair. There are no complications.

Prognosis.—Unfavourable as regards cure; fairly favourable as regards duration.

The main aids to prognosis in such a case are the following :—

1. Whether the special lesion is of the nature of fibroid induration or excavation.

The former is much more favourable than the latter.

2. The state of the digestion, pulse, weight, and temperature.

3. The occupation and mode of life.

4. The prospect of adequate and persevering treatment.

These cases sometimes go on indefinitely, enjoy fair health, and perhaps do arduous work, but they do not recover. Change from an unsuitable to a suitable occupation is a matter of great importance.

In the long run, these cases usually succumb to some form of pulmonary disease, not always to the direct effects of tuberculosis, but perhaps to an intercurrent pneumonia, pleurisy, or bronchitis.

CASE IV.—Tubercular disease of the pneumonic type has progressed rapidly to partial excavation of one apex, and has then undergone arrest. The general condition is fair, but the patient is thin, rather short of breath, and incapable of severe exertion.

Prognosis.—Doubtful ; a minority of these cases get on well ; in the majority of cases the extension of the disease to the rest of the lungs is only a question of time.

It will be remembered that Jaccoud held this type of case within certain limits to be especially favourable.

CASE V.—Excavation exists at one apex, infiltration of a portion of the same lung, slight signs at the opposite apex.

Prognosis.—Unfavourable, but duration may be very prolonged.

Temperature, weight, strength, digestive capacity, prospects of thorough and persevering treatment must always be accorded their due weight. These cases are practically incurable. A duration of over twenty years has been known to me.

CASE VI.—Supervening upon an old chronic lesion, tubercle has become rapidly disseminated throughout a large area of both lungs. The temperature is high, and there is much wasting and prostration.

Prognosis.—Highly unfavourable ; death probably in a few weeks.

In a certain small proportion of such cases the acute process becomes arrested, and the case lapses into the chronic category.

CASE VII.—Phthisis has supervened upon typhoid fever, measles or whooping-cough. It has assumed the sub-acute caseating form.

Prognosis.—Unfavourable.

These cases usually do badly. In exceptional instances, the active symptoms of tuberculization abate, and the case takes on one of the ordinary types of chronic fibro-caseous phthisis, to which the broad rules of prognosis, already sufficiently commented upon, may be applied.

I put forward the foregoing types of disease, and the prognostic indications which they suggest, fully realizing their very limited value. Nevertheless, they may afford the practitioner a certain small amount of guidance. Cases of phthisis are so numerous, and the prognosis varies within such wide limits that we cannot rest content

with a vague general prognosis, whether of the pessimist type of past years or of the optimist type of to-day. We must approximate to rules which will enable us to say with some approach to accuracy in any given case :—

- (a) Whether there is a reasonable hope of complete recovery.
- (b) Whether the case is, upon the whole, favourable, a good rally probable, and treatment likely to repay its cost in time and money.
- (c) Whether the case is, upon the whole, unfavourable and admitting of only a moderate degree of improvement.
- (d) Whether the case is definitely unfavourable and admitting only of a slight degree of palliation.
- (e) Whether the case is obviously hopeless and systematic treatment useless.

The economic aspects of these questions often come before us. Patients, or their friends, say with much reasonableness, "If I make a great sacrifice in time and money, spend a prolonged period in a sanatorium or at a health resort, can you give me any assurance that the result will be commensurate with the expense and trouble?" No doubt, a positive reply cannot always be given, and the patient's means, social position and domestic circumstances may all require to be carefully weighed. But an answer of some sort to this natural and reasonable question—the best that we can arrive at—should be given.

It hardly needs insisting, in concluding our survey of this subject, that in a disease where the unexpected so often happens, where the casual and the unforeseen play so large a part, where the patient's wisdom, courage, and self-control count for so much, our prognosis should always

be guarded and a dogmatic attitude should in general be avoided.

It will often, however, be the duty of the practitioner to tell the patient's friends candidly that the case is hopeless, the end only a question of time, and that sanatorium or other treatment will be futile.

LECTURE IX.

THE TREATMENT OF PULMONARY PHTHISIS.

SUMMARY.

Preventive Treatment.

Measures of general hygiene, disposal of sputum, notification, teaching of hygiene in schools.

Specific Treatment.

History of the use of tuberculin, personal experience of the remedy, the new tuberculin, Koch's rules for its administration.

Antiseptic Treatment.

- (a) Inhalations of thymol, creosote, formaldehyd.
- (b) Intra-tracheal injections of menthol.
- (c) Intravenous injections of formalin.
- (d) Internal administration of creosote.
- (e) Electrical treatment.

Hygienic Treatment.

History—Its leading principles, results, views of Weber, Morin, Burton-Fanning, Snow, Turban.

Question of "cure"; whether cure or marked improvement is lasting or not; criteria of improvement; drawbacks to sanatorium treatment; Huggard's views.

IN this and the following lecture, I shall deal with the treatment of phthisis.*

I can treat this large question only in a very summary way, and I wish to be understood as giving in the main the results of my own experience rather than attempting

* For a fuller discussion of the modern treatment of phthisis, see Transactions of the British Congress on Tuberculosis, Encyclopædia Medica, Art. Tuberculosis, by R. W. Philip; Pulmonary Phthisis, by Percy Kidd in Allbutt's System of Medicine, and the writings of Brehmer, Dettweiler, Fraenkel, Weber, Powell, Theodore Williams, Allbutt, Ransome, West, Osler, Trudeau, Knopf.

to do justice to the views of others. I shall, however, at least, mention every form of treatment, of the value of which I am personally convinced. The multitude of remedies which have been extolled in phthisis is, for the more part, a measure of their inutility. Nevertheless, great progress has been made in the treatment of the disease in modern times, and the present outlook is more hopeful than it has ever been before. We approach the task of arranging the treatment in any case of phthisis of a fairly favourable type, with the well-founded hope that something—perhaps much—can be done. The process of educating the public to a just measure of what may be fairly expected from treatment, and what is clearly beyond its range, is in progress, and when that process is further developed, the task of the medical practitioner will be much lightened.

Preventive Treatment.

As to the supreme importance of prophylactic measures there can be no second opinion. Phthisis is easily preventable, but only at the cost of considerable forethought, trouble, time, and money.

No child, however infected the stock, must be regarded as the necessary victim of tubercle.

No family, however ravaged by the disease, must be regarded as inevitably doomed to further ravages.

The ultimate victory of science and civilization over tuberculosis is certain.

The preventive treatment of phthisis includes the following departments—

A. Measures of general hygiene.

(a) Better housing of the lower classes.

(b) Attention to cleanliness, ventilation, disposal of domestic refuse.

- (c) Draining of damp subsoil, removal of filth, proper sewerage.
- (d) Attention to the food supply, to milk and to water.
- (e) Regulation of mills, factories, warerooms and workshops.
- (f) Care of the young, viz. attention to the hygiene of schools, the provision of crèches and playgrounds.
- (g) The inculcation of temperance.
- (h) The provision of adequate air-spaces in large cities.

B. The disposal of sputum ; the disinfection of the rooms occupied by consumptives.

C. Compulsory notification.

To enter fully into the above points would be to write a manual upon public health. I must content myself with emphasizing a few considerations.

The housing of the working-classes is the most fundamental fact of all in connection with the prevention of phthisis. That the disease spreads rapidly in small, dirty, ill-ventilated and overcrowded houses, is the outstanding fact regarding phthisis. Reform must begin—has, indeed, already begun—at this point.

There is no necessity to adopt the language of panic in connection with this matter. We can teach truly that the consumptive is not necessarily a source of danger to his household, provided that necessary precautions are taken—precautions not excessively onerous or costly, but that carelessness involves serious risks. We should teach that the infective agency of tuberculosis is comparatively weak, comparatively easy of control, only capable of active

dissemination where conditions favourable to its growth and activity prevail,—those conditions being essentially overcrowding, filth, and bad air. In clean, airy rooms, with strict care regarding the disposal of the sputum, the danger of infection is practically *nil*.

Of the immense importance of disinfecting the rooms occupied by consumptives there can be no question. The cases which have come under my observation of phthisis following the occupation of infected rooms especially by young people admit of only one explanation. It is our duty to instruct the public that to permit a healthy individual, especially a child or adolescent, to occupy a room recently inhabited by a consumptive, without adequate disinfection, is a crime.

Care as regards ventilation and the disposal of sputum is now becoming general. This is a great gain.

Compulsory notification of phthisis should certainly be advocated. Such a step is a necessary preliminary to grappling with the question of prevention on a large scale by the public-health authorities. The provision of municipal sanatoria for the disease would necessarily follow compulsory notification.

The facts regarding the prevention of phthisis should form part of the science teaching in our schools. The time is past for regarding preventive medicine as a mysterious matter, to be exclusively reserved for experts. While the popularizing of medical treatment is to be discouraged on the broad and clear ground that successful treatment depends upon accurate diagnosis, and that accurate diagnosis presupposes a trained observer, no such objection applies to the popularizing of the principles of preventive medicine. These principles are, in the main, simple and intelligible, quite within the mental compass of an intelligent boy or girl, and it is our duty to make them

an integral part of the intellectual outfit of every youth who passes through our schools.

The question of the relation of milk and meat to tuberculosis is still unsettled. The most acute divergence of view prevails on this subject, and I cannot here enter into the evidence. While awaiting decisive evidence on this important matter, we are certainly justified on general grounds in insisting upon the thorough inspection of dairies and the control of the sale of meat by municipal authorities. It is to be feared that neither of these duties is at present very efficiently discharged in this country at least.

I pass on to consider the general treatment of phthisis. I shall deal with the subject under the following heads:—

- (a) Specific treatment.
- (b) Antiseptic treatment.
- (c) Hygienic treatment.
- (d) Symptomatic treatment.
- (e) Climatic treatment.

Specific Treatment.

I need not repeat the story of the discovery of tuberculin, by R. Koch, in 1890; of its general adoption as a therapeutic agent in phthisis; and of the disappointment of the hopes which had been raised so high.

I should like to speak with much reserve on this subject, as my experience of the original tuberculin was very small, and I have not tried the new tuberculin at all. In view of the opinions, not only of Koch, but of Spengler, Turban, Krause, Heron, McAll Anderson, Goetsch, Osler, Rembold, Kirchner, and others, it would appear that we may have to reconsider our judgment regarding these remedies. It was my good, or evil, fortune in my earliest trials with tuberculin to encounter a case in hospital practice

where the injurious effects of the remedy were patent. A quiescent and favourable case of phthisis was converted by a few injections of tuberculin into an active and progressive one, new foci of disease appeared in the lungs, and I confess that my therapeutic ardour was daunted. Since then, I have never given tuberculin as a therapeutic agent. It must be admitted that at the time of which I speak, viz. 1890, the rules and restrictions now laid down for the administration of tuberculin were not generally understood. Koch now teaches that this medicament should only be used in early and afebrile cases, and that the dose should at first not exceed from one-tenth of a milligram to a milligram. Regarding the use of tuberculin in early, afebrile and uncomplicated cases, he says, "That tuberculin exercises an exceedingly favourable influence on all such cases, and even completely cures them as a rule, is a fact of which I have repeatedly convinced myself."* In view of such confident language, it is disappointing to find one of the most recent English authorities expressing himself as follows: "Clinical observation, however, has demonstrated that the remedy has unfortunately by no means the value which Koch originally claimed for it. Even in the case of lupus, where the effect is most striking, and the conditions most favourable for cure, there has been no case of complete cure, while in phthisis the results have been utterly disappointing. Even in the most successful cases, the improvement has not been greater than is often seen, under various other forms of treatment, when patients are taken into hospitals and carefully tended." In many instances the disease runs its course unaffected, and in others it is aggravated."†

There seems at present no satisfactory evidence that

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 95.

† S. West: Diseases of the Organs of Respiration, vol. ii. p. 541.

the new tuberculin is either free from the dangers of, or of greater curative potency than, the old.

For the present, I must content myself with saying that my experience of tuberculin is small and unfavourable. Of the value of tuberculin for purposes of diagnosis there is, of course, no doubt, and the evidence that one or more minimal doses given for this purpose are harmless is apparently conclusive.

I subjoin Koch's latest rules for the administration of tuberculin:—

1. Only patients that have no fever, and in whom the process has not advanced too far, are suited for the treatment.

2. One begins with a very small dose, and increases it so slowly, that only very slight reactions, or even none, take place.

3. If reactions take place, tuberculin must not be injected again until the temperature has been normal for one or several days.

4. The treatment with tuberculin must be repeated till, after an interval of three to four months, the capability of reaction is permanently extinct.*

Antiseptic Treatment.

The antiseptic treatment of phthisis may be conceived as contemplating the following objects—

(a) The destruction of the tubercle bacilli *in situ*, or the limitation of their activity by remedies reaching the part *per viam respirationis*.

(b) The destruction of the bacilli, or the limitation of their activity by remedies acting through the blood.

(c) The immunization of the tissues, so as to render them resistant to the action of the bacilli.

* *Loc. cit.*

These ends are sought to be achieved in the following modes—

- (a) By inhalations of creosote, carbolic acid, thymol, or formaldehyd.
- (b) By intra-tracheal injections of menthol or izal.
- (c) By intravenous injections of formalin or protargol.
- (d) By the internal administration of creosote or guaiacol.
- (e) By the introduction of antiseptic medicaments to the seat of the malady through the pores of the skin by means of static high currents of electricity of high and medium tension (Crôte method).

I may say at once that my own somewhat extensive trial of antiseptic treatment in phthisis has been limited to two of the above methods—viz. antiseptic inhalations and the internal administration of creosote and guaiacol. I must confess that I am not enthusiastic about either of these methods. That inhalations of some medicaments (*e.g.* formalin) have a favourable influence over the secretions from bronchi or *vomicæ*, and may tend to check some of the secondary infections (*e.g.* streptococci, staphylococci, pneumococci, influenza bacteria), which are so common in phthisis, is highly probable. That they exercise any direct controlling influence upon the bacilli of tubercle seems doubtful. Our knowledge of the life history of these organisms and their powers of resistance to chemical substances, suggests serious doubts whether any such substances can be brought into direct contact with the foci of the disease so as to destroy the bacilli without seriously injuring the lung substance.

A prolonged trial of inhalations of creosote, carbolic acid, thymol, and other such remedies, fully convinced me of their futility.

Intra-tracheal injections, and intravenous injections,

have at least the merit of courage, not to say audacity. That injections of menthol in olive oil or of izal in glycerine into the trachea ever reach the foci of disease seems dubious. The bronchial tree is complicated ; in such a labyrinth it would seem a matter of chance whether remedies of this class ever reach their destined mark. Intravenous injections of formalin, I confess, suggest various dangers to my mind.

Creosote and guaiacol have now had a prolonged trial in phthisis. Their use was evidently suggested by the hypothesis that these agents might in some way assist to antagonize the action of tubercle bacilli, render the tissues more resistant, perhaps promote fibroid change. A large number of observers of the highest eminence have expressed themselves as favourably impressed with the results of this treatment. I confess that, after a long trial of these remedies, I have seen nothing to justify the high place which they have held in the treatment of phthisis. I am inclined to suspect that creosote and guaiacol have shone by reflected light. Their general adoption as remedies in phthisis was almost coincident with the wide recognition of the value of systematized hygiene in this disease, and the better results obtained in recent years have been sometimes attributed in part to medicaments which, I believe, have had little real efficacy. No doubt, creosote sometimes exercises a favourable influence upon gastric derangements. That it, in any sense, renders the lungs or the tissues generally immune to the action of bacilli is a most doubtful proposition. In recent years, I have gradually discarded creosote and guaiacol—reluctantly, because I could not ignore the weight of evidence in their favour—but I feel certain that the results of treatment have been at least as good without, as with, these remedies. The systematic use of remedies of very doubtful utility is to be

deprecated. Patients are so ready to take our drugs, so unready to accept our directions as regards wholesome living. The systematic use of such remedies as creosote and guaiacol in phthisis seems likely to divert the attention of both patient and doctor from measures of real utility. I would suggest to those of my medical brethren who set much store upon the use of creosote and guaiacol in phthisis, that they should make a temporary trial of hygienic and tonic measures without the aid of these remedies, and to abide by the result. I assure them that they may make the experiment with a perfectly easy conscience.

The Hygienic Treatment of Phthisis.

Writing in the year 1856, the late Dr. Henry MacCormac, of Belfast, said: "It is impossible to urge in terms too strenuous, too explicit, the indispensableness of open-air life and effort in respect of the prevention of disease in general, and of phthisis in particular."

MacCormac's view of the causation of phthisis was, that it was due to breathing "re-breathed" air. His pathology was erroneous, or at least inadequate—Koch's discovery was still a quarter of a century in the future—but his views on treatment were sound, and in some degree original. The modern hygienic treatment of phthisis is simply an elaboration and methodizing of MacCormac's views. Still, it must be admitted, that greater credit is due to those who make important truths actual and operative than to those who divine them by a process of intuition, but fail to make them practically available. Brehmer and Dettweiler were true pioneers, and it is not without justice that the hygienic treatment of phthisis is known in Germany as the *Brehmer-Dettweilerische Behandlung*. This

treatment has now had a trial of thirty or forty years, though only recently upon a large scale, and it is not premature to ask what its results have been.

My own experience of the treatment has been considerable, and has had relation to sanatoria in Ireland, England, Germany, Switzerland, and America.

It may seem something of an anachronism to argue the case for a therapeutic method so well established as the sanatorium treatment of phthisis, but I still find, on the part of many practitioners, a considerable degree of scepticism as to its value. I am often confronted with unfavourable statistics, and I find the view sometimes expressed that this line of treatment is only a passing fashion in medicine, and not a genuine advance in therapeutics. My own view is that the hygienic treatment of phthisis (of which sanatorium treatment is simply a systematized mode) is a method, sound in principle, not difficult of application in practice, and of genuine utility, if, only, we do not expect too much from it. It rests upon the sound principle that, in the absence of a trustworthy specific for phthisis, the best means of combating the malady is to raise the general health to its highest power—flood, as it were, the tuberculized area with healthy blood and pure air—in the conviction, that when these conditions are fulfilled, the disease will in many cases abate. In well-nourished and well-ventilated lungs, it is not rash to assert, tubercle maintains itself with difficulty. Whether the patient will do better in an organized sanatorium or in his own extemporized and solitary shelter, how much medical supervision counts for, how far certain auxiliary lines of treatment and the control of symptoms influence the final result, what weight is to be attached to locality, climate, telluric conditions—these are interesting and important questions, but I think we should teach that the absolutely indispensable

conditions of the hygienic treatment of phthisis are few and simple. They are these—

That the patient shall breathe absolutely pure air at all times, day and night ;

That he shall take abundance—even an excess—of simple, nourishing food ;

That he shall follow certain well-recognized rules as regards rest and exercise ; and

That he shall dispose of his sputum so as not to re-infect himself.

This is the essence of the case, though by no means all. The choice of food, the improvement of digestion, the regulation of the bowels, the hygiene of the skin—these are points of real importance. I repeat, however, that the essentials of the hygienic method are few and simple, that they may be applied anywhere and by anybody, with suitable guidance, and that if this treatment is to become universally available it must be simplified, rather than elaborated. I have seen the most admirable results when the patient has simply built himself a common wooden shelter on his father's farm, lived on milk, eggs, and such-like food, and made it a rule to rest when his temperature was above normal. No doubt, we must allow for the influence of the personal equation. Not every patient can be trusted to manage his own case ; many imperatively require supervision and control, but this is not because the treatment involves any real difficulties. It is because system, intelligence, and perseverance are so often lacking on the part of the patient.

As regards the results of sanatorium treatment, we desire information on the following points :

- (a) What are the general results of the treatment in the different stages of phthisis, adopting the time-honoured, if somewhat unsatisfactory,

classification of the three stages, infiltration, softening, excavation?

- (b) In what proportion of cases can we fairly use the word "cure"?
- (c) When arrest of the disease has been achieved, is this arrest permanent or transient?
- (d) When great improvement has been obtained, is the maintenance of that improvement mainly dependent upon the patient's habits, occupation, and mode of life?
- (e) Are there any cases unsuitable for sanatorium treatment?
- (f) Are there any serious drawbacks to sanatorium treatment?

Some of these questions do not at present admit of a precise or dogmatic answer.

As regards the general results of treatment in the different stages of phthisis, I may adduce the following evidence:—

Sir Hermann Weber (in a private letter to myself, dated July 17, 1903) gives the following results in the case of patients treated in sanatoria in and out of England:—

1st stage—cured, 74 per cent.

2nd stage—cured, 48 per cent.

3rd stage—cured, 19 per cent.

Dr. Turban * (Davos) reports—

1st stage—97 cases; improved, 95; not improved, 2.

2nd stage—205 cases; improved, 174; not improved, 31.

3rd stage—106 cases; improved, 56; not improved, 50.

* K. Turban, Beiträge zur Kenntniss der Lungen-Tuberkulose. Wiesbaden, 1899.

In 116 cases the sputum was free from bacilli at the time of the patient's discharge. 14 patients (3·4 per cent.) died while under treatment.

Capacity for exertion (*Leistungsfähigkeit*) on discharge from the sanatorium was noted as unimpaired in 80 of the 95 cases of improvement in the 1st stage, in 76 of the 174 cases of improvement in the second stage, and 5 of the 56 cases of improvement in the 3rd stage.

Dr. Burton-Fanning* reports—

716 cases collected from various sanatoria in England, Scotland, and Ireland.

Quiescence of the disease or relative recovery was obtained in 37·4 per cent.

Amelioration, 40·2 per cent.

No improvement, 22·3 per cent.

A gain in weight took place in 92 per cent.

A precise classification of the above cases according to stage and extent of lung mischief was not available.

Only 52 cases (7·4 per cent.) could be described as "cases of slight lung mischief." Of this group 46 (*i.e.* 88·4 per cent.) "did well, and obtained relative recovery with arrest of the disease." Tubercle bacilli disappeared from the sputum in only 19 of these 46 patients (*i.e.* 41·3 per cent.). In some of the remaining cases the presence of bacilli was doubtful.

Dr. Morin† (Leysin, Switzerland) reports—

1st stage—cured, 50 per cent. ; improved, 40 per cent.

2nd stage—cured, 10 per cent. ; improved, 67 per cent.

3rd stage—cured, 3·2 per cent. ; improved, 48 per cent.

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 199, et seq.

† *Ibid.* p. 229.

Dr. W. V. Snow (Bournemouth) reports—

In the early stages apparent arrest of the disease in 63 per cent.

In the intermediate stages apparent arrest of the disease in 40 per cent.

In the advanced stages apparent arrest of the disease in 11 per cent.

Dr. Snow adds, "the percentage of cases of arrest would be increased if numbers did not leave at the end of three months to resume their occupations." *

It would be easy to add more evidence of the same description. In spite of considerable discrepancies in the above statistics, we may note a considerable degree of harmony on some important points, viz.—

- (a) That a large degree of improvement, amounting in many cases to complete arrest or "relative cure," may be expected from sanatorium treatment.
- (b) That the degree of success attained has a close relation to the stage of the disease at which sanatorium treatment is undertaken.

My second question, viz. In what proportion of cases can we fairly use the word "cure"? is a very difficult one to answer.

Now, by "cure" in this connection we must understand not an absolute *restitutio ad integrum*, which is undoubtedly a very rare event, but rather the complete restoration of the general health, with abeyance of all symptoms, restoration of average working power, and locally the signs of a limited and quiescent lesion. The practical point of interest is, In what proportion of cases can we fairly speak of "cure" in this qualified, but important, sense?

We have seen that "cure" is claimed by some

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 215.

authorities in a proportion varying from 37 to 74 per cent. of cases, the percentage being notably higher in cases where treatment is undertaken in the first stage.

My own experience of sanatorium treatment, while highly favourable as regards such results as "great improvement," "relief of all urgent symptoms," "restoration to fair general health," does not incline me to speak very confidently on the subject of "cure," though I should hardly be inclined to endorse West's blunt statement: "Sanatorium treatment cannot change the natural course of the disease; if this is expected, disappointment will follow. It will not cure the disease, but it will assist the patient to resist the inroads of it." *

In this connection, the very embarrassing question arises, How often does cure take place in phthisis without treatment, or where a diagnosis of the disease has never been made? The proportion of persons dying of other diseases whose lungs present evidence of healed tuberculosis is not less than 10 per cent., and may be nearer 20 per cent. I think it highly probable that in many of these cases the disease never progressed to the extent of causing local signs, but this is theoretical. I have no doubt in my own mind that if we make a point of examining the sputum in every case of "lingering cold," or repeated "bronchial attacks," we shall sometimes find tubercle bacilli when not in the least expected, and that in some of these cases the disease does not proceed farther. If this form of arrest occurs without treatment in a certain important proportion of cases, *a fortiori* it may be expected to occur in connection with sanatorium treatment. "Cure," taking the term *simpliciter*, is an expression to be used with strict economy in connection with the results of treatment in phthisis.

* Diseases of the Organs of Respiration, vol. ii. p. 565.

On the other hand, great improvement, arrest of the disease for a longer or a shorter period, control of all active symptoms, are frequent results of sanatorium treatment. In the absence of definitely unfavourable symptoms, such results may probably be expected in more than half of cases in the first stage, in considerably less than half of cases in the second stage, and in only a small minority of cases in the third stage.

When "arrest of the disease" or "great improvement" is obtained, are these results lasting?

Of all questions in this connection this is the most important. The treatment of phthisis on a large scale is an economic question. Granting that great benefits may be obtained from sanatorium treatment, are these benefits permanent or at least fairly enduring? Are they worth their cost? On the answer to these questions probably turns the whole attitude of civilized nations towards the sanatorium treatment of phthisis. Directors of sanatoria should make a rule not only to tabulate the results obtained during the period of treatment, but to follow the after history of their patients.

Turban* supplies the following important evidence with regard to this question:—

To the interrogatory, "Has the patient's condition remained stationary, improved, or got worse since his discharge from the sanatorium?" 225 replies were received. In 68 the condition was described as "stationary," in 127 as "improved," in 30 as "worse." In 126 cases the patients were found to be dead. The cases were spread over a period of seven years. The length of time which had elapsed from the patient's discharge from the sanatorium until the date of inquiry, viz. 1897, is not stated.

* *Loc. cit.*

Turban's results, therefore, work out as follows :—

Total number of patients in one septennium	408
Obtained marked improvement	325
Died while under treatment	14
Died before 1897	126
Improved after discharge from sanatorium	127
Stationary since " " " ...	68
Lost ground since " " " ...	30

In reply to the question, Is the patient's activity (*Leistungsfähigkeit*) "not impaired," "slightly impaired," "much impaired," or "annulled," the replies numbered 226, and afford the following information :—

Not impaired	151 cases
Slightly impaired	45 "
Much impaired	18 "
Annulled	12 "

These results are, upon the whole, encouraging. They point to the conclusion that the improvement obtained by sanatorium methods is not a mere transient rally, but possesses some elements of permanence. It must be borne in mind that a patient usually leaves a sanatorium a thorough convert to hygienic methods; he is fanatical for fresh air, has sound views upon food, rest, exercise, sputum disposal; he knows the dangers of certain occupations. Hence, it is not over sanguine to hope that the benefits which he has obtained may endure. He has started upon a new career, and may have a new fate. It is important to emphasize these points, as a widespread prejudice exists that the improvement obtained in sanatoria is brief and illusory.

The criteria of a patient's improvement are chiefly the following :—

- (a) Gain in weight, strength, capacity for exertion, breathing power, pulse.
- (b) Freedom from pyrexia.
- (c) Diminution or disappearance of physical signs.
- (d) Disappearance of tubercle bacilli from the sputum.

Gain in weight, taken alone, is a fallacious test of improvement. Rest and high feeding will produce a temporary gain in weight with most patients, even if no general improvement is really in progress. Some most melancholy examples have occurred in my experience of patients who, in response to hyper-alimentation, have gained several stones in weight without any real or lasting improvement. In these disappointing cases, in spite of the gain in weight, the patient is weak, short of breath, his pulse is unduly frequent, and the physical signs in the lungs do not recede. The prognosis in such cases is unfavourable. On the other hand, a steady and progressive gain in weight, if accompanied by improvement in pulse, energy, and local physical signs, is most important.

Freedom from pyrexia is found in nearly all cases which make really satisfactory progress. A slight degree of afternoon pyrexia is not inconsistent with marked amendment.

Retrocession of the physical signs is highly important. In favourable cases crepiti become fewer and drier, or perhaps disappear altogether. Signs of shrinkage of the affected apex appear. Compensatory emphysema of the unaffected lung may be detected.

Disappearance of the bacilli from the sputum is of favourable augury, but bacilli may persist in the sputum of patients whose general condition is quite favourable.

As regards the questions whether there are any cases unsuitable for sanatorium treatment, or any serious drawbacks to sanatorium treatment, we may consider

these two cognate problems together. I do not know that I can do better than quote the remarks on these subjects which Dr. W. R. Huggard addressed to the British Congress on Tuberculosis (1901):—

“Is a sanatorium the best place for every case? For some cases, yes; but certainly not for all. For all those people who are unable otherwise to secure suitable attention, hygienic arrangements and medical supervision, a sanatorium is obviously the best place. It is also the best place for the wilful, the thoughtless, and the impulsive. The great advantage of sanatorium life is that the patient is drilled into the practice of hygiene, and his task is made easier by having fellow-learners in the same discipline. He is made to take sufficient food, to rest and to exercise according to his condition; and he is more likely to have answered his daily prayer not to be led into temptation. In the sanatorium, too, the patient is under the immediate eye of the doctor; and the beginnings of new ailments are seen at once. Patients suffering from laryngeal tuberculosis are, also, as a rule best treated in a sanatorium, where their tendency to talk too much can be kept under control. In many cases, however, the cast-iron discipline of a sanatorium is unnecessary, in many others unavailing. Where the needful hygienic and general conditions can be secured with the intelligent co-operation of the patient, all the requirements of treatment can be carried out without recourse to the rigid system of a school. In some cases, also, it must be recognized that one ending only to the disease is inevitable. When this is the case, little is to be gained by drilling the patient. Discipline is out of place, and, except occasionally, the thermometer does not then afford information to compensate for the anxiety or the inconvenience occasioned by its use. . . . The psychological element is important in the treatment of tuberculosis. . . .

One great object of treatment in most cases will be to place the patient where there will be the greatest freedom from worry, fretting, irritation, depression, or excitement. This object may or may not be best secured in a sanatorium."*

In concluding this part of my subject, I should like to enter a *caveat* against two common features of sanatorium treatment, viz. forced over-feeding and the frequent taking of rectal temperatures. That patients must be exceedingly well fed, even to a point which has hardly a parallel in any other disease, may be freely admitted; but there is a limit, and that limit has, I fear, often been passed. The theory that dyspepsia may be ignored in this connection, and that forced feeding may be made an almost invariable rule, does not commend itself to me.

Four-hourly rectal temperatures are quite unnecessary. They subserve no really useful end, and tend simply to create or aggravate a most objectionable form of morbid introspection.

It is sometimes laid down that if a patient must ultimately earn his living in his own country he should be treated in a home sanatorium. I do not think this point deserves the importance which has been attached to it. Some of the best cases of prolonged arrest which I have known have followed a residence in the Alps.

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 224.

LECTURE X.

THE TREATMENT OF PULMONARY PHTHISIS (concluded).

SUMMARY.

The Symptomatic Treatment of Phthisis.

Measures to improve digestion and nutrition, to promote the hygiene of the skin, to control pyrexia, hæmoptysis, cough, pain, night-sweating ; the management of complications.

The Climatic Treatment of Phthisis.

Question as to the direct or indirect influence of climate.

Classification of climates :—

1. The oceanic climate, its general characters, advantages and disadvantages of sea-voyages, cases of phthisis for which the sea-voyage is suitable, contra-indications.
2. The marine climate, its many varieties—The Riviera, British resorts, Canary Islands, Madeira, Algiers, Tangier, Southern California.
3. The inland climate of plain or plateau—Egypt, Southern Algeria, South Africa, Colorado, Australia, New Zealand.
4. The mountain climate—meteorological characters, indications and contra-indications, results of treatment at high altitudes ; views of Williams, Weber, Stephani, Turban.

Types of consumptive patients, and indications which they afford for the choice of climate.

The Symptomatic Treatment of Phthisis takes at the present day a very minor place.

If the broad principles of hygienic treatment have been adopted, nothing can be worse than a fussy attention to symptoms. Nevertheless, the phthisical patient is the subject of many conditions which can be favourably influenced

by remedies, and no objection can be raised to such treatment, if its strictly subordinate character is steadily borne in mind.

I shall consider these remedies under the following heads:—

- (a) Remedies to improve digestion and nutrition.
- (b) Remedies to promote the hygiene of the skin.
- (c) Remedies to control pyrexia.
- (d) Remedies to control hæmoptysis.
- (e) Remedies to control cough and pain in the chest.
- (f) Remedies directed against complications, *e.g.* ulceration of the larynx, ulceration of the intestines, amyloid degenerations.

(a) Our first thought—and sometimes our last—in the treatment of phthisis should be the promotion of nutrition.

The digestive condition of the consumptive is variable. Often he suffers much from dyspepsia; not seldom he surprises us by digestive vigour. The dyspepsia of phthisis takes on many forms, and cannot be usefully differentiated from dyspepsia of other origins. In these days of high feeding in phthisis, the catarrhal type of dyspepsia (*e.g.* subacute gastritis) is not uncommon.

Tuberculous ulceration of the oral cavity and aphthous stomatitis are occasionally observed. They are usually found in severe and advanced cases, and are of very evil augury.

The tongue varies much. If pale and flabby, with slight fur, atonic dyspepsia is probably present. If thickly coated, with prominent papillæ, gastric catarrh may be suspected. If red, raw-looking, or glazed, intestinal ulceration may be conjectured.

These conditions must be treated on general lines. It is well to make sure that the food is not excessive in

amount or unsuitable in character. It is no infrequent experience of mine to be informed by patients that they consume two or more quarts of milk and four to six eggs daily. Yet such patients sometimes complain sadly that "they never have any appetite." It would be strange if they had. In the dyspepsia of phthisis the drugs most often useful are alkalies and bitters, especially nux vomica. The bowels often require attention. The moderate use of aperients is unobjectionable. Intestinal ulceration, when it arises, is usually most intractable. Opium is the best remedy, and a diet of peptonized milk or of raw-beef juice or raw-beef sandwiches may be tried. Rest should be enforced.

Tonics and artificial nutrients have a considerable utility in phthisis. Vegetable bitters, nux vomica, quinine, arsenic, and the hypophosphites may sometimes be employed with advantage.

Cod liver and maltine are valuable nutrients.

Alcohol should be used with a sparing hand.

(b) Remedies to promote the hygiene of the skin.

With the adoption of hygienic methods, night-sweating usually ceases to be troublesome. The skin should be sponged over once or twice daily with tepid water, to which methylated or rectified spirit or eau de Cologne should be added. This is a most valuable procedure.

Atropin has a potent influence upon night-sweating, but in the long run it does more harm than good. I have abandoned its use, except in the most exceptional cases.

The routine application of counter-irritants to the chest must be strongly condemned. Where pleurisy is present, or where there is much pain in the chest, the occasional and purely temporary employment of vesicants may be approved, but under all other circumstances the cardinal

rule in phthisis should be to keep the skin of the consumptive scrupulously clean. There are few sadder sights in practice than that of the consumptive's chest plastered over with iodine, and in a state of positive filth—a sight, unhappily, by no means rare. It can only be called barbarous.

The clothing should be warm, but not heavy. All forms of “chest protectors” are to be avoided. The patient should sleep in some light woollen material.

(c) Remedies to control pyrexia.

Pyrexia commonly yields to rest, fresh air, regulated diet, and attention to the hygiene of the skin. Antipyretics, such as antipyrin, phenacetin, salipyrin, salicylate of soda, have quite failed in my hands to do any permanent good. They should certainly be used sparingly, and only for certain definite ends. Quinine often does good, but rather from its tonic than its antipyretic properties. The pyrexia of phthisis is often due to mixed infections. Antiseptic inhalations may be tried, but the results are seldom brilliant.

(d) Remedies to control hæmoptysis.

The essential points in the management of hæmoptysis are rest, quiet, abstinence from conversation, as little food as possible and that of the simplest kind, morphia or opium, saline purgation.

Most of the astringents commonly employed are probably inert or hurtful.

Turpentine has sometimes succeeded well with me.

Ice to the surface of the chest is sometimes useful.*

(e) Remedies to control cough and pain in the chest.

The broad rule in phthisis is to treat the constitutional

* For a full discussion of the causes and treatment of hæmoptysis, see Lecture XI.

state and ignore the cough. Sometimes, however, the cough is so obtrusive, bringing on vomiting after meals or disturbing sleep, that some treatment is necessary. The routine administration of cough mixtures is, of course, one of the most baneful of errors.

Cough may be justifiably the subject of treatment under the following conditions:—

- (a) When it is irritative, incessant, with little expectoration, and interfering with digestion or sleep.
- (b) When it is probably due to ineffectual efforts to expel viscid mucus from a cavity.
- (c) When it is due to excessive bronchial secretion.
- (d) When it is due to laryngeal ulceration.

In the first class of case, morphia, hydrocyanic acid, bromides and Virginian prune are the best remedies.

In the second class, alkalies, given in hot water or hot milk, are useful.

In the third class we may try inhalations of tar, benzoin, or formalin (2 to 3 per cent.).

In the fourth class we may use sedative inhalations—benzoin, conium, hop; or antiseptic sprays; or powders, such as iodoform.

For the relief of pain in the chest, small flying blisters or a liniment of menthol and chloroform may be tried. This latter combination is a valuable one.

(f) Remedies directed against complications.

Finally, we have to consider the treatment of certain complications. In laryngeal ulceration the general treatment is the chief point. The local treatment consists in the use of antiseptic or sedative remedies in the form of inhalations, sprays, powders, solutions. A very active treatment of the local condition is seldom advisable. The ulcers should be kept clean.

The treatment of ulceration of the intestines has been already considered.

Fistula in ano requires the attention of the surgeon. Syphilis may require specific treatment. It is generally taught that in tubercular subjects anti-syphilitic treatment should not be very active.

Change of Climate in Phthisis.

With the general recognition of the preponderating influence of hygienic measures, habits, and mode of life in the treatment of phthisis, the relative importance of locality and climate has in recent years somewhat receded. Yet these factors cannot be ignored. It is alleged by some observers that "weather" has little or no influence upon the course of phthisis, that patients do as well in winter as in summer, in bad seasons as in good. This is not my experience. I cannot doubt that weather counts for something with the consumptive: affects his nutrition, as well as his cough and his breathing; affects him psychically as well as physically. If, as is often alleged at the present day, climate has no influence upon phthisis—direct or indirect—then it follows, as a necessary inference, that to the consumptive, sunshine and sunlessness are indifferent, tonic air and relaxing air, shelter and exposure, wind and calm. Will any one accept this doctrine thus nakedly expressed? No doubt, we cannot attribute to climate any definite, specific, antagonizing influence upon phthisis, but interpreted in a broad sense, so as to include all meteorological and telluric influences, it is a factor which only a very one-sided pathology can ignore. Many years ago I wrote as follows on this subject: "Climatic treatment is not a complete therapeutics, and will be only a snare if so interpreted. It is a means to an end, not an end in itself; a powerful adjunct to hygienic and medicinal measures,

not a substitute for them ; a channel of escape from vicious habit and abnormal mode of life, not a mysterious remedy or an unfailing specific."* I see no reason to alter these views. I am entirely unable to understand the views of those who hold that locality has no influence in the treatment of phthisis, one of the most outstanding facts in my experience being that patients do well in one locality and do ill in another, although the general conditions of life remain much the same. I may add that this is practically the unanimous opinion of patients themselves.

Climate may be usefully regarded from two points of view :

- (a) First, as regards its direct influence upon the patient's pulmonary condition, his nervous system, or his nutrition.
- (b) Secondly, as regards its indirect influence in either facilitating or impeding open-air life, hygienic measures, healthful occupations.

These two factors are sometimes difficult to disentangle. No doubt, the second is often the more important of the two. Yet I cannot doubt that the first is also significant, however it may elude the attempt to give it precise scientific expression. That many tubercular patients do well at home is a fact—a most happy fact—to which we have in recent years become increasingly alive. But what shall we say about those who do ill at home? Do they, or do they not, make any better progress at Davos, San Remo, in the South African veld, upon the uplands of Queensland, in Colorado, in Egypt? Let those answer who have really a large experience in this matter. Some tubercular patients will do well anywhere, provided they adopt a rational mode of life: Some tubercular patients

* J. A. Lindsay : *The Climatic Treatment of Consumption*, p. 226. 1887.

will do ill anywhere, no matter how they live. Is there, or is there not, an intermediate class, to whom locality and climate are factors which may incline the scale towards, or away from, recovery? I believe there is. Experience must finally settle the question.

The question of climate confronts us also in relation to the complications of phthisis. Bronchial catarrh, anæmia, nervous irritability, sleeplessness, dyspepsia, laryngeal ulceration may be present, and it will hardly be maintained that to such conditions the temperature and humidity of the air, the presence or absence of local shelter, the tonic or relaxing properties of the atmosphere are questions of indifference.

Again, we find that while successful sanatoria may be founded under the most diverse conditions of locality and climate, yet local conditions have not been entirely ignored in the selection of their sites. A dry subsoil, an agreeable outlook, a free circulation of air combined with local shelter, a maximum of sunshine and a minimum of fog and mist—such are some of the conditions which are commonly aimed at.

Moreover, in this connection we have to consider the case of the patient who, having undergone a course of sanatorium treatment and secured arrest of his malady, desires advice as to the best localities for either temporary or permanent residence. We are asked the question whether the recrudescence of the disease is to be feared if life is resumed in the former locality, or whether a change would be advisable.

On all these counts—and the subject might easily be further developed—it will not be found convenient or advisable to act upon the assumption that locality and climate are matters which can be safely ignored in the management of phthisis. It is most desirable not to overstate the

importance of climate and climatic change. It is equally essential not to under-estimate their importance.

Climates are many. Health resorts are innumerable. In their choice, true indeed is it that "experience is fallacious and judgment difficult." *

We may fairly ask the question, Have the climates found useful in phthisis any common characters? Have Davos and St. Moritz any *commune vinculum* with Egypt and the Orange Colony, San Remo and Mentone with Colorado and Queensland, Algeria and Sicily with Bournemouth and Ventnor? The answer often given to this question is that climates are useful in phthisis simply in proportion as they permit the patient to spend a longer or a shorter time in the open air with comfort and satisfaction. This somewhat simple formula hardly seems to exhaust the subject. The patient's age, constitution, temperament, the stage and activity of his disease, the complications which may be present, will be found to count for something in relation to locality and climate. One of the wisest of the ancients has said, "Those things which we use most and oftenest have the greatest influence on health; and water and air are of this nature." † The favourite climates for phthisis are, for the most part, sunny, without excessive heat, presenting long spells of continuous favourable weather, suitable for outdoor exercise, free from violent winds, of moderate or low grade of humidity. On the other hand, no climate is generally recommended which involves much continuous bad weather, frequent high winds, excessive humidity, sunlessness.

The patient's temperament—lymphatic, neurotic, bilious, sanguine (vague as these terms are, and difficult of scientific application, there can be no doubt that they point to profound differences)—has much to say with regard

* Hippocrates : Aphorism I.

† Aristotle : Politics, vii. 2, 4.

to his response to climate influence. This is probably even a more fundamental point than the stage or activity of his disease. We shall return to this question in connection with the various forms of climate.

The limits of these lectures only permit a very summary treatment of the large subject of climate and phthisis. I wish to give in the briefest form the results of a considerable experience in this department. I shall treat the subject from two points of view, viz.—

- (a) The leading types of climate, their meteorological features, advantages and disadvantages, indications and contra-indications.
- (b) The leading types of phthisis, and the indications as regards climate and locality which they afford.

There will be a certain convenience, if also some unavoidable repetition, in this twofold mode of considering the subject.

The climates recommended in phthisis may be grouped under the following heads:—

1. The oceanic climate, such as a patient enjoys during a long sea-voyage.
2. The marine climate, *e.g.* Bournemouth, Ventnor, Torquay, Rostrevor, Queenstown, Mentone, Bordighera, San Remo, Alassio, Rapallo, Jersey, Arcachon, Biarritz, Sorrento, Palermo, Malaga, Algiers, Tangier, Canary Islands, Madeira, Southern California, Florida, Tasmania.
3. The inland climate of plain or plateau, *e.g.* Egypt, Pau, Montreux, Meran, Algeria (Hammam R'Ihra, Biskra), South Africa (the Karoo, Griqualand West, Orange River Colony, the Transvaal, Natal), Colorado, California, parts of New South Wales, Queensland, and New Zealand.

4. The mountain climate, *e.g.* Davos, Clavadel, Arosa, St. Moritz, Pontresina, Maloja, Wiesen, Montana, Leysin, Arolla, Les Avants, Caux, Andermatt, the Adirondacks, the Andes, the Carpathians.

The above classification is one of practical convenience only, and will not stand strict criticism. It is open to several objections, *e.g.* that the elevation of some of the resorts in the third class is equal to, or greater than, those in the fourth class.

1. *The oceanic climate.*

The leading features of this climate are the following : Equability, a high average of humidity, a high degree of purity and free circulation of air, much ozone.

The best sea-voyage is the all-round voyage to New Zealand *viâ* the Cape of Good Hope, and home *viâ* Cape Horn. It involves about twelve weeks at sea. No voyage of shorter duration than this can be expected to do much good in pulmonary cases.

The popularity of the sea-voyage as a remedy in phthisis has fallen off decidedly in recent years, and it is worthy of note that this change in medical practice has coincided with the gradual effacement of the old clipper-ship and the substitution of the modern steamship. That this change, however convenient from other points of view, has been a loss to the invalid, I make no doubt. The old-fashioned sailing-ship, with its roomy cabins, its leisurely life, its absence of excitement, its slower progress through different latitudes, its longer duration, was more suitable to the invalid than the crack liner with its swift speed, its rapid progress through different degrees of latitude, its often crowded cabins, its gayer life, its briefer duration.

The sea-voyage combines a very perfect form of physical and mental repose, continuous fresh air without

fatigue, an agreeable sense of passive movement, life on a new plane. On the other hand, the ventilation of the cabins is not always good, the food is sometimes of mediocre quality, there is an element of possible boredom and ennui, and the discomforts of continuous bad weather are sometimes serious. I entertain no doubt, however, that, granting a well-found, well-ventilated ship, good cuisine, agreeable society, and reasonable luck in weather, a sea-voyage is a powerful restorative in many conditions of debility, and has an important, but very limited, application in phthisis. The typical case for a sea-voyage is that of a young man (20 to 30) who has become infected with tuberculosis as the result of study, continuous indoor occupation, or the struggles of commercial or professional life, whose general health is not gravely impaired, and who is fond of the sea and attracted by the prospect of travel. Such patients, it may be observed, often shrink from the tedium and rigid routine of sanatorium life, but readily acquiesce in the suggestion of a sea-voyage.

The sea-voyage is also useful in the so-called "strumous" cases, where a sluggish pulmonary lesion co-exists with affections of bones or joints.

The sea-voyage is unsuited for all cases of advanced disease and all cases with active symptoms. Pyrexia, marked debility, much dyspepsia, laryngeal and intestinal complications, all contra-indicate the sea-voyage. Hæmorrhage is not a contra-indication. A certain amount of nervous irritability is rather an indication for the sea-voyage, provided we can secure for the patient a good ship, a comfortable cabin, and a wise and congenial companion. Some writers have laid much stress upon the noises and distractions of ship-board life, upon the crash of the engines, the roar of wind and wave, the bustle of the saloon. My own recollections, based upon a not inconsiderable

experience of the sea, are rather of placid days, much fine weather, calm mental and physical, a life of repose and freedom from irritation, a life sometimes indeed dull and monotonous, but peaceful and restful. Insomnia is rarely complained of on shipboard after the first few days, seasickness soon abates in nearly all cases, appetite and digestion are commonly active, weight in most cases increases. There are, however, exceptions to these rules, and sometimes it is clear that the patient has an intolerance of sea life.

Dr. Theodore Williams recommends sea-voyages also in cases of chronic cavity, where the tubercular disease is unilateral and quiescent, and adds that in strumous phthisis he has repeatedly seen cavities contract and a gain of weight of from one to two stones occur during a sea-voyage.* I believe this is so, but it is difficult to say how cavity cases will do on shipboard.

A point of some importance in connection with sea-voyages is the management of the patient after landing. Indiscretions at this stage often undo the good obtained during the voyage. The patient should be warned that on landing he must exercise special care as regards over-fatigue, exposure, errors in eating or drinking. He must not make too abrupt a change in his habits.

In recommending a sea-voyage, age, sex, and tastes must have weight. Upon the whole, the young do decidedly better at sea than the middle-aged or old, men better than women, and those who have a natural taste for the sea better than those who have not. It is not well to advise a sea-voyage for a patient who has a natural dislike for the sea.

2. *The marine climate.*

Marine resorts form a large class, and present funda-

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 7.

mental variations as regards (*a*) temperature, (*b*) humidity, and (*c*) wind-exposure and shelter.*

Most of the favourite marine resorts are characterized by warmth, moderate humidity, and some degree of shelter. The Rivieran resorts (San Remo, Mentone, Bordighera) represent the type of dry marine resorts. Algiers and Palermo represent the type of moderately moist marine resorts. Madeira and the Canary Islands represent the type of decidedly moist marine resorts. Dry marine stations may be reckoned as "bracing" or "stimulating." Moist marine stations may be reckoned as "relaxing" or "sedative."

The Rivieran resorts have lost something of their former popularity, in part owing to the competition of rivals, especially the mountain sanatoria, but in part, also, to their inherent defects, especially as regards windiness and instability of temperature. Yet the winter, in a favourable season, is often very charming. Long spells of mild, sunny, gently stimulating and fairly calm weather are not uncommon.

For the consumptive, Mentone and San Remo are, perhaps, the best of the Rivieran resorts. Where quiet is desired, Bordighera may be tried. Grasse sometimes does well, but is apt to be rather cold. Where economy is a consideration, Alassio or Rapallo may be recommended. Nice, Cannes, and Monte Carlo are less suitable for the invalid.

Visitors to the Riviera must be advised that the climate demands watchfulness, that sudden changes of temperature, especially just after sun-down, are common, that the combination of bright sun and cold winds is somewhat

* Dr. Gordon, of Exeter, has recently brought forward a good deal of evidence to show that in Devonshire exposed districts have more phthisis than sheltered districts.

treacherous, and that clothing and habits must be arranged accordingly. It is a matter of common complaint that visitors to the Riviera do not submit themselves to medical control as much as might be wished. The atmosphere, traditions, and *milieu* of the Riviera are favourable to pleasure-seeking and enjoyment rather than to continuous, watchful, and serious treatment. Monte Carlo acts as a disturbing influence. These are serious drawbacks.

According to Dr. Theodore Williams,* the cases most suitable for Riviera are the following :—

- (a) Phthisis, in which inflammatory processes have played a large part in predisposing to the disease.
- (b) Strumous phthisis.
- (c) Laryngeal phthisis.
- (d) Unilateral tuberculization rather than bilateral.
- (e) The large class of consumptives who, either from extent of disease, or feebleness of circulation, or advancing years, are unable to endure the rarified atmosphere and cold of the high altitudes. Most of these patients love warmth and cannot take enough exercise, when the thermometer is below zero, to maintain it.

The Riviera is, in my experience, generally unsuitable for neurotic patients, for active disease, and for cases where irritable dry bronchitis is present. The *eretische constitution* does not do well in the Riviera.

The Home Resorts—Ventnor, Bournemouth, Torquay, St. Leonards, Dawlish, Sidmouth, Salcombe, Ilfracombe, Rostrevor, Queenstown, Glengarriff—come under the class of marine stations, and suit many cases well for winter residence.

They are appropriate for cases of phthisis which for various reasons—medical, financial, personal, or domestic—

* *Loc. cit.*

decide not to go abroad. The results obtained sometimes compare favourably with those obtained at more distant stations. Some patients shrink from the fatigue of a long journey, some are repelled by the idea of foreign customs and a strange cuisine, others dislike separation from friends. No doubt these objections would be trivial if the advantages of foreign resorts were always striking and decisive. But this is far from being the case. Ventnor, Bournemouth, and Torquay are good winter stations, and may safely be recommended in many cases. They are especially suitable for cases of chronic phthisis, where digestive troubles are prominent, for patients who are reluctant to leave their own country, and for cases where there is more than the usual amount of uncertainty regarding the patient's prospects and progress. Advanced cases may obtain the stimulus which change of air and scene affords with a minimum of fatigue and risk by selecting one of the home sanatoria.

The Canary Islands have a remarkably mild, equable, and sunny, but somewhat damp, climate. The winds usually blow from the African mainland, and are somewhat hot. Orotava, La Laguna, Güimar, Santa Cruz (Teneriffe), Santa Cruz (La Palma), Las Palmas (Grand Canary) may be mentioned among the available resorts in these islands. There is much complaint of the frequency of diarrhœa and bowel complaints amongst visitors, but some of my informants assure me that these troubles are mainly attributable to indiscretions with regard to food, especially eating excessively of fruit or eating fruit of bad quality.

My personal trial of the Canary Islands in cases of phthisis has not been large, but it has been fairly favourable. These islands are worthy of recommendation in certain cases of phthisis where anæmia and debility are

marked, where irritable bronchitis is present, or when the patient craves warmth and sunshine—also in advanced cases where palliation of symptoms is the only object contemplated.

Madeira is, upon the whole, less desirable than the Canary Islands. My experience of it is small and unfavourable. The most suitable cases for this resort are cases of catarrhal phthisis, cases of phthisis in the elderly, cases where palliation is all that we can hope for.

The Sicilian resorts—Palermo, Catania, Taormina—belong to the same class as the Canary Islands, and may be tried in similar cases.

Algiers has a good winter climate, with a high proportion of fine weather, moderate humidity, and much sunshine. The hygienic condition is not good, and dust is often troublesome.

Tangier has a somewhat similar climate, but is more under the influence of winds from the Atlantic. It is not a suitable resort for consumptives. The sanitation is oriental—a point which is in itself decisive.

There are some good resorts upon the coast of Southern California, *e.g.* San Diego, Santa Barbara. In North California, Monterey is the chief resort.

The coast of Florida is a favourite resort with patients from the northern states.

3. *The inland climate of plain or plateau.*

The resorts enumerated under this category present many variations as regards elevation and temperature. In the main they are dry, sunny, and tonic. The marked diurnal oscillations of temperature which are found in most of these resorts do not seem to be disadvantageous, provided they occur with regularity, and can, therefore, be reckoned with. Such oscillations have a tonic influence.

Egypt has great attractions for the patient of ample means who can command every luxury, and whose condition does not preclude him from travelling about and enjoying the scenery and the antiquities. Such cases, are however, exceptional, and Egypt is not well adapted for ordinary cases of phthisis. In spite of the magnificent climate and in many places excellent accommodation, the conditions of life and the social *milieu* are not suitable for consumptives. The Nile voyage, however charming, has a very limited utility for phthysical cases. Dust is apt to be troublesome in Egypt. The season is short, as patients cannot stay with advantage when March is past. The choice of locality in Egypt lies between the following places: Cairo, Helouan, Mena House, Luxor, and Assouan. Cairo should be avoided by the consumptive. The hygiene is bad, and the social atmosphere unsuited to such cases. I have made only a very limited trial of Egypt in phthysical cases, and I give the foregoing opinions with some reserve. Dr. Sandwith records that "after eighteen years of life in Egypt he has only as yet seen one case of a European living in the country becoming tubercular."* He admits, however, that the natives suffer largely from the disease. I should be inclined to limit the recommendation of Egypt to early and quiescent cases, especially in middle-aged patients where bronchitis or weak circulation or rheumatism is present, and in cases where the historic and antiquarian attractions of the country were likely to prove of special interest.

The same remarks might apply, *mutatis mutandis*, to the interior of Algeria. Hammam R'Ihra is a good spring resort, but is not very suitable for winter. Biskra, on the edge of the Sahara, is recommended for cases where desert air and quiet are desired.

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 33.

South Africa has earned a reputation in phthysical cases, and I have made a somewhat extensive trial of it. The best parts of it are the high plateaus of the interior, especially certain districts of the Karoo, Griqualand West, Orange River Colony, the Transvaal, and Upper Natal. The coast regions are, upon the whole, unsuitable. Amongst the places which are recommended for phthysical cases the following may be enumerated: Cradock, Beaufort West, Matjesfontein, Graaf Reinet, Aliwal North, Bloemfontein, Ladybrand, Harrismith, Kimberley, Heidelberg (Transvaal), Greytown (Natal). Many patients, however, wisely avoid the towns, and seek quarters upon a farm—usually a Dutch farm. The mining towns are dusty, and in every respect undesirable. The climate presents many variations, but is in the main dry and sunny, with a good deal of atmospheric rarefaction, and with hot summers and cool, rather than cold winters. In many parts of the interior the winter is practically rainless. Drought is often troublesome, dust-storms are frequent, and sanitation is primitive. The accommodation is rather poor, and the food-supply leaves a good deal to be desired. My experience of cases sent to South Africa has been rather conflicting. I have had results quite beyond my expectations. I have had not a few disappointing failures. I entertain no doubt that the climate is, in the main, a very good one if the patient gets well into the interior, but the journey thither is fatiguing, and it is not always easy to secure an environment favourable for treatment. The best type of case for South Africa is an early case of phthisis in a young man who does not object to "rough it," who is in fair general health, and who looks forward to permanent residence in the country, if the results as regards health are satisfactory. Cases with much bronchial secretion, some emphysema, rheumatism,

or slight valvular mischief may be sent to South Africa. Advanced or active cases should on no account be sent, and, as living is expensive, it is indispensable that the seeker after health should be adequately provided with financial resources. The fate of poor patients with advanced disease, who go out to South Africa under the vague impression that the climate will work miracles, is usually a deplorable one.

Colorado is a good type of the "inland plateau" type of climate. Denver, Colorado Springs, and Manitou Springs are among the best-known resorts. The climate is dry, sunny, and tonic. The cold in winter is sometimes severe, and cold winds are apt to be annoying. The Colorado climate is, from some points of view, intermediate between that of the South African plateaus and the Alps. Dr. Denison of Denver assures us that "tuberculous infection reaching an advanced stage of the disease is so rare an occurrence in Colorado as to be remarkable."* My own trials of Colorado have been limited to a few cases, but I have been well pleased with the results. Patients speak enthusiastically of the climate, accommodation is good, and convalescent cases can find employment. Colorado may be recommended in the same types of case as South Africa.

Los Angeles and Pasadena in Southern California may be reckoned as inland resorts, but from the climatic point of view they are akin to the resorts upon the Californian seaboard.

Australia has long had a repute in pulmonary disease, a repute which is somewhat on the wane, but which has still a certain foundation. The climate is in many places a magnificent one for nine months in the year—dry, sunny, tonic, and exhilarating, with a high average of fine days,

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 25.

and affording exceptional opportunities for outdoor life. On the other hand, the summer season is in many places quite too hot, and the dust-storms which sometimes prevail at this season are only too likely to have a disastrous effect upon the consumptive. On the Riverine plain of New South Wales, where the climate for a large part of the year has great charm, the thermometer not uncommonly rises in summer to 105° , 110° , or even 120° in the shade, dust-storms are not infrequent and destroy all vegetation, and drought is sometimes prolonged and disastrous. It is impossible to approve of such conditions for the consumptive. Among the districts which are specially recommended for consumptives may be mentioned, Armidale (New South Wales), Beechworth (Victoria), Macedon (Victoria), Orange (New South Wales), Toowoomba (Queensland), Mount Victoria (New South Wales), Bathurst (New South Wales), Goulburn (New South Wales), Maranoa (Queensland). The coast-line of Australia is, in general, unsuitable for consumptive cases. The Australian climate is in a high degree tonic and stimulating. It is therefore best suited to quiescent cases and to "lymphatic" constitutions. It is unsuited to all irritable conditions, to neurotic patients, and to active disease.

Life on a sheep-farm in certain parts of Australia presents many advantages, and it may be possible for the invalid to escape the summer heats by resorting to the mountains or to Tasmania. A consumptive should never be sent to Australia with the view of engaging in indoor work in one of the large Australian cities. Advice to this effect runs counter to the most elementary principles of the treatment of phthisis.

New Zealand has a milder, more humid, and more windy climate than Australia. There are, naturally, great

variations amongst the different parts of the islands. On the whole, the climate is not particularly well suited to consumptive cases, though some patients undoubtedly do well there. The best districts are, perhaps, Napier and the Canterbury Plain.

4. *The mountain climate.*

Sir Hermann Weber gives the following catalogue of the meteorological features of the climates of elevations exceeding 3,500 feet.

- (a) Diminished atmospheric pressure; diminished density or greater rarity of air.
- (b) A lower degree of absolute and relative humidity of the air.
- (c) Absence or great infrequency of mists.
- (d) Greater transparency of the air.
- (e) Greater diathermancy of the air.
- (f) Lower shade temperature.
- (g) Greater difference between sun and shade temperature.
- (h) Greater purity of the atmosphere from organic and inorganic particles. Absence or rarity of microbes. The presence, probably, of more ozone in the air.
- (i) Comparatively little wind (in winter).*

The conditions of life at the high-altitude resorts are a little difficult of realization by those who have no practical experience of them. In favourable weather heat in the sun may be so strong as to call for sunshades, while in the shade icicles may be forming. Though the shade temperature may be many degrees below freezing point, the cold is little felt, unless there be wind. Exercise is freely taken, amusements are numerous, life is by no means dull.

* The Mineral Waters and Health Resorts of Europe, p. 352.

The proportion of bright sunny days is high—though the seasons vary much in this regard—and when the weather is at its best it is absolutely perfect. For persons who can bear the cold and the rarefaction of the air, the climate is in a high degree tonic and bracing, nutrition is in many cases successfully stimulated, nervous energy is promoted, the number of red corpuscles in the blood is increased,* the expansion of the chest is augmented.

Whether cold *per se* is desirable for consumptives is difficult to say. It seems probable that cold is inimical to bacillary activity, but in the case of man the question is much involved owing to its relation to habits, occupation, and nutrition. If a cold climate involves confinement to the house, or interferes with the food-supply, it will be *pro tanto* injurious to the consumptive. It is worth recalling that Commandant Peary reports that several members of his crew in his last Arctic expedition (1902), who were slightly consumptive, obtained much benefit from their stay in the Arctic Regions.† He is of opinion that some day sanatoria will be established in those regions for consumptives! In all probability, the patient with a fair degree of circulatory and digestive vigour, and capable of taking adequate exercise will do better in a cold climate than in a warm one.

The high altitudes may be tried in most cases of phthisis, provided certain well-understood contra-indications are absent.

These contra-indications are chiefly the following:—

- (a) Circulatory weakness, whether due to organic cardiac disease or not.
- (b) Much bronchitis and emphysema.
- (c) Albuminuria.

* Viault, Egger, Kündig, Mercier, and others.

† I quote from a report in the *Times*, but I have forgotten to note the date.

(d) Rheumatism.

(e) Marked nervous irritability.

(f) Inability to take exercise and to withstand cold.

The best results are obtained in young male adolescents with incipient lesions, in hæmorrhagic cases, and in cases consequent upon pleurisy.

It is generally thought that cases of laryngeal ulceration are unsuitable for high altitudes, but Turban and Huggard both report a considerable degree of success in the treatment of such cases at Davos. On the whole, it seems better to send these cases to a warm and rather dry climate.

No stage of the disease precludes resort to the high altitudes, but naturally the early cases do best. Some cavity cases, if the lesions are not extensive and the general circulatory, digestive, and nervous condition satisfactory, do fairly well at Davos, Arosa, or St. Moritz. A moderate degree of pyrexia is not a contra-indication. Dr. Theodore Williams reports that resort to the high altitudes led to arrest of the disease in 58 per cent. of his cases, and to improvement in 87 per cent. In excavation cases arrest occurred in 21 per cent. of cases, and great improvement in 61 per cent.*

Dr. Turban reports that of 408 cases treated in his sanatorium at Davos, good results were obtained in 325 cases (76·6 per cent.), while in the remaining 83 (20·4 per cent.) the results were unfavourable.†

Dr. Stephani reports that of 150 cases treated at Montana, complete cure was obtained in 12 per cent., improvement in 50 per cent., arrest in 20 per cent., and no benefit in 18 per cent.‡

* *Loc. cit.*

† Beiträge zur Kenntniss der Lungentuberkulose, p. 134, etc.

‡ Transactions of the British Congress on Tuberculosis, vol. iii. p. 80.

Sir Hermann Weber finds "that the effect of these climates—*i.e.* of the high altitudes—had been better than that of any other climatic group or sea-voyages."*

My own experience of the high-altitude stations is in accord with that of Sir Hermann Weber.

Patients may go to the high-altitude sanatoria at any time of the year, but the winter is the best season. There seems no foundation for the idea that special dangers attend the melting of the snow. A short stay at some intermediate station, on leaving the high altitudes, seems usually advisable.

Vigilant medical supervision is especially obligatory at the high-altitude stations. The patient's habits and amusements must be strictly controlled.

I shall now select some well-known clinical types of phthisis, and inquire what can be said as regards the class of resort most likely to be found useful in their treatment. It must be understood that the suggestions given possess only a very limited validity, inasmuch as every case must be considered upon its merits, and personal idiosyncrasies cannot be ignored.

TYPE I.—The common case, where incipient phthisis is suspected but positive proof is lacking.

Slight cough, perhaps an occasional trace of blood in the expectoration, some loss of flesh, some degree of languor and debility—such is a common group of symptoms. There are no physical signs in the chest, and sputum is either absent or bacilli are wanting.

Probably a large proportion of these cases are tubercular, but we may not be able to advance beyond the point of suspicion. Much judgment is required to avoid

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 22.

the opposite errors of premature panic and disastrous procrastination.

It is questionable whether these cases should be sent to a sanatorium. They are usually most reluctant to go ; constant association with more or less advanced cases of phthisis is of doubtful propriety when a doubt rests upon the diagnosis, and the psychical effect of sending mere "suspicious" cases to such institutions is not free from objection. On the other hand, such patients readily welcome, as a rule, the suggestion of climatic change. Often their condition is due to over-pressure of some sort, and the release from arduous duties or the escape from an unfavourable environment may be an essential element in the treatment.

The choice of climate in such cases is obviously rather wide. A long sea-voyage, a winter in the Alps, a few months in South Africa, may in different cases be advised. I have usually advised either the first or the second alternative, and I have seen excellent results from both. The state of the circulatory and digestive organs, and of the nervous system, will assist to differentiate between them. If such cases are sent to the Alps, it is well to select some resort—*e.g.* St. Moritz—not much frequented by advanced cases of phthisis.

TYPE II.—A case similar to the foregoing, but with definite, though slight, signs in the lungs, or bacilli in the sputum.

Sanatorium treatment should usually be preferred in such cases.

If, for any sufficient reason, sanatorium treatment is not decided upon, the choice will lie between the high-altitude stations and such inland resorts as South Africa or Colorado.

Some of these cases do very well at the home stations. The sea-voyage is, in most cases, inadvisable.

TYPE III.—A case where phthisis sets in suddenly with profuse hæmorrhage in the midst of apparently good health, the physical signs being slight, and the constitutional state satisfactory.

A period of complete rest and treatment at home will obviously be necessary in the first instance.

These cases do very well at the high altitude stations. I have also seen excellent results from a long sea-voyage. Sanatorium treatment is sometimes advisable.

TYPE IV.—A case where phthisis has supervened upon pleurisy or broncho-pneumonia, and where "inflammatory" conditions may be supposed to have played a considerable part.

These cases do well at the high altitude stations.

They also do well in the Riviera.

The state of the circulation, digestion, and the nervous system will determine the choice between these two types of climate.

TYPE V.—A case where phthisis has set in with active catarrhal symptoms.

These cases should be regarded with caution, as well-marked catarrh may point to widespread tuberculization.

These cases do not get on well in the mountains or at sea. They do better on the south coast of England, in the Riviera, the Canary Islands, Egypt, or Algeria. If we judge that, owing either to the bronchial or the nervous condition the case requires stimulation, the Riviera or Egypt may be selected. If we judge that the case requires soothing, we may select the Canary Islands.

TYPE VI.—A case presenting laryngeal complications—irritative or ulcerative.

Many of these cases should be kept at home.

Irritable laryngitis is sometimes benefited by a sedative climate, *e.g.* Bournemouth or the Canary Islands. Laryngeal ulceration sometimes does well at the high-altitude sanatoria, but more generally should be advised to select a moderately dry climate of low elevation, *e.g.* the Riviera.

TYPE VII.—A case characterized by albuminuria or associated with rheumatism or heart disease.

A dry, warm climate should be selected.

The Riviera, Egypt, or Algeria may be chosen.

The home sanatoria, the long sea-voyage, and the high-altitude stations are unsuitable.

TYPE VIII.—A case where excavation has occurred at one apex, but the disease is limited and the general state fair.

These cases do fairly well in many climates, but rarely recover completely. The high-altitude stations are usually best, but the inland stations, the long sea-voyage, or the home resorts may sometimes be advised.

TYPE IX.—A case of extensive lesion and steadily progressive disease, with more or less active symptoms.

These cases should usually be kept at home.

If a change be thought advisable, one of the English stations may be selected.

Madeira or the Canary Islands may sometimes be chosen with a view to the palliation of symptoms.

TYPE X.—A case of the so-called “strumous” phthisis, *i.e.* where disease of bones or glands is present in addition to pulmonary lesions.

A bracing marine resort is the best for these cases. Hastings, Margate, San Remo may be selected.

The long sea-voyage is sometimes useful.

TYPE XI.—A case of limited disease, where dyspeptic symptoms are prominent or where diarrhoea is present.

These cases often do best at one of the home resorts.

TYPE XII.—A case where phthisis has supervened upon typhoid fever, influenza, or measles.

In the case be recent, and it is still doubtful whether its course is going to be subacute or chronic, these cases should be kept at home, or may be sent to one of the English resorts.

If it is clear that the case is going to pursue a chronic and fairly favourable course, and the constitutional state is good, these cases may be sent to the high-altitude resorts, or the dry marine resorts, or the inland plains.

Speaking generally, the main guides to the selection of climate in phthisis are the activity of the morbid process and the constitutional condition of the patient. The stage of the disease is less important. The extent of the disease is, however, a consideration of weight. The more extensive the disease the less likely is the patient to react favourably to tonic climates, *e.g.* those of high altitudes.

Often our most difficult task is to decide whether climatic change should be tried at all or not. If this question be decided in the affirmative, we have to determine whether our aim is the arrest and cure, or only the palliation, of the disease. Then, we must consider whether the patient is likely to respond to an actively stimulating, moderate stimulating, or sedative climate.

These questions may not in every case admit of a

definite answer, but they are not impracticable or useless questions.

There is no necessary conflict between sanatorium treatment and climatic treatment. The two may be successfully combined. We have instances of this combination in the well-known sanatoria at Davos, Montana, Leysin, and many others.

Dr. Burney Yeo says most truly, "What the consumptive patient most needs is a combination of climate and sanatorium treatment; for the patient, if left to his own devices, may make bad use of a good climate, while with skilful guidance in a sanatorium, he may make good use of a bad one. Care without climate is better than climate without care." *

* Transactions of the British Congress on Tuberculosis, vol. iii. p. 20.

LECTURE XI.

THE CAUSES AND MANAGEMENT OF HÆMOPTYSIS.

SUMMARY.

Table of the causes of hæmoptysis.

Search for cause in any given case.

The so-called "bleeding from the throat," its usually fallacious character.

Hæmoptysis from heart disease.

Alleged cases of hæmoptysis in healthy people, the so-called "arthritic hæmoptysis."

Hæmoptysis in pulmonary phthisis—frequency, influence of age, family history, type of disease, stage of disease, amount of bleeding, immediate causes of bleeding, prognostic import of hæmoptysis.

Dangers of hæmoptysis—(a) Immediate, (b) Remote.

Question of "hæmorrhagic phthisis."

Physical characters of the blood.

Fallacies in the differential diagnosis of hæmoptysis.

Hæmoptysis from blood dyscrasiæ.

Some of the rarer forms of hæmoptysis.

Treatment of hæmoptysis.

HÆMOPTYSIS is a frequent and important symptom.

The patient commonly exaggerates its immediate dangers; the physician rightly attaches weight to its diagnostic significance.

The causes of hæmoptysis may be enumerated as follows:—

1. Pulmonary tuberculosis.

(a) Hyperæmia of bronchial mucous membrane.

(b) Ulceration of a bronchiole.

- (c) Rupture of an aneurism upon the pulmonary artery.
- 2. Valvular disease of the heart, especially mitral stenosis.
- 3. Pulmonary conditions other than tuberculosis.
 - (a) Pulmonary infarction.
 - (b) Capillary bronchitis.
 - (c) Plastic bronchitis.
 - (d) Emphysema.
 - (e) Gangrene.
 - (f) Pneumonia.
 - (g) Bronchiectasis.
- 4. Certain blood conditions.
 - (a) Scurvy.
 - (b) Hæmophilia.
 - (c) The anæmias, including leuco-cythæmia.
 - (d) Purpura.
 - (e) Malignant types of the specific fevers.
- 5. Degeneration of the pulmonary vessels.
 - (a) Bright's disease.
 - (b) The so-called "arthritic" hæmoptysis.
- 6. Aortic aneurism.
- 7. Epithelioma of the œsophagus, carcinoma of the lungs.
- 8. Vaso-motor disturbance, *e.g.* hysteria.
- 9. Actinomycosis.
- 10. The endemic hæmoptysis of Japan and Korea, due to the *distomum pulmonale*.
- 11. Traumatism.

Of the so-called "vicarious" hæmoptysis I have no experience.

We shall first consider the general diagnostic problem usually involved in a case of hæmoptysis, and then enter into the examination of details.

There is a definite history of hæmoptysis. How shall we proceed to determine the probable cause?

Often the cause will be self-evident. The patient may present clear signs of phthisis, or mitral disease, or of scurvy, hæmophilia, or some of the anæmias. Such causes as pulmonary infarction, whooping-cough, capillary bronchitis will usually be evident from the history and symptoms. Aortic aneurism and bronchiectasis have their own clinical picture, and will not often give rise to serious difficulty.

In a case of hæmoptysis, without evident cause and with no characteristic history, we may say, with a high degree of probability, the patient has either phthisis, or heart disease, or some blood dyscrasia.

As regards that frequent statement of patients, "I had an attack of spitting of blood, but the doctor said it came from the throat," it may be laid down with confidence that such a statement is hardly ever well-founded. It generally means that, no physical signs being discovered in the lungs or heart, and no evident source for the bleeding being recognizable, it has been assumed that the blood came from the throat. Now, I think it may be affirmed that free bleeding from the throat is not common under any conditions. It may occur in advanced laryngeal ulceration, but such cases are usually clear. There is generally a definite history of tuberculosis, a poor constitutional state, hoarseness or aphonia, sometimes a certain degree of dysphagia. Again, blood may come from the naso-pharynx and pharynx in a variety of conditions. But such cases rarely involve any serious difficulty. The pharynx or nasal passages may be seen to be congested or ulcerated, the blood may come in part from the nose, and, if we have the opportunity of inspecting it, we shall probably find it dark in colour, watery, and mixed with saliva or nasal mucus.

Here, again, we may lay down a useful working—if not invariably accurate—rule. If the nasal passages and pharynx are fairly healthy in appearance, if no marked congestion or ulceration be present, if no varicose veins can be seen, then it is highly improbable that the seat of the bleeding is in these regions. The hæmoptysis of heart disease is not, as a rule, difficult of detection. It is not at all a common symptom in early cases with good accommodation. It occurs chiefly in mitral stenosis and mitral regurgitation, more often in the former lesion, generally in cases where the signs of heart disease and passive congestion are gross and evident, and its recognition is usually a simple matter. We may remark that the association of valvular disease of the heart and phthisis being a rare one, the discovery of a cardiac cause for hæmoptysis makes the co-existence of pulmonary tubercle improbable.

If, then, no obvious explanation of an attack of hæmoptysis is forthcoming, if we can exclude heart disease and blood dyscrasias, what is the probability that phthisis is the cause of the hæmorrhage? That probability is a high one. Nearly all apparently uncaused attacks of hæmoptysis, nearly all attacks where no other obvious morbid state co-exists, are in my judgment due to pulmonary tuberculosis. This may seem a somewhat sweeping statement, but I believe it will bear the test of experience. No doubt many of these patients get well and never develop the clinical symptoms of phthisis, but then we have to bear in mind that evidence of healed tuberculosis is found in at least 10 per cent of the lungs of persons dying of all causes. I have been for some time in the habit of attaching much weight to a history of hæmoptysis in a case where the diagnosis of phthisis was on various grounds doubtful, and I have found it a point of great value. We must, of course, make sure that the alleged attack of hæmoptysis

was a genuine one, not a mere streak of blood in the sputum occurring after violent coughing—to which but little weight can be attached—nor an attack of bleeding from the gums, pharynx, œsophagus, or stomach. I believe we can usually exclude these sources of fallacy by attention to the story of any intelligent patient. In genuine hæmoptysis the blood is usually coughed up, and there is a history of cough preceding the hæmorrhage, the blood is more or less mixed with air, its colour varies much and is not to be relied upon, but it is often bright and “arterial,” the amount of blood lost varies within the widest limits, the bleeding, if at all free, does not stop abruptly but a smart hæmorrhage is followed by more or less blood-spitting for hours or days, some rise of temperature frequently follows the hæmoptysis. Of these various points, the one which gives us most assistance when we are analysing a patient’s story is the persistence of slight blood-spitting after a sharp hæmorrhage. If a patient informs us that upon a certain date he had a sharp hæmorrhage, accompanied by cough and followed by a gradually diminishing amount of blood for hours or days, we may conclude with much confidence that the blood came from the lungs, and if heart disease, blood dyscrasias, and other obvious morbid states are absent, we shall not be often wrong in concluding that tuberculosis is present.

Are there any cases of recurring attacks of hæmoptysis in persons otherwise healthy where the attacks are really of no importance ?

Such cases are described by most writers upon the lungs, and it is undeniable that some persons suffer from periodic attacks of hæmoptysis but remain in good health and never exhibit any definite pulmonary signs. The late Sir Andrew Clark believed he could recognize a variety of

hæmoptysis occurring in persons of the "arthritic diathesis" and not due to any definite pulmonary lesion. I have met with some cases of recurring hæmoptysis in persons otherwise apparently healthy, but I suspect most of them are due either to slight tubercular deposit in the lungs or obscure valvular lesion of the heart. That sound and healthy lungs ever bleed recurrently is, I think, a most doubtful proposition. It is dangerous in practice ever to make light of hæmoptysis, or to assume that it may be due to any constitutional peculiarity, involving no serious issues. The absence of physical signs and the total abeyance of symptoms go for nothing as regards the presence of pulmonary tubercle. It is certain that this condition may exist without either signs or symptoms.

We shall now consider the chief points relating to hæmoptysis, due to tubercular disease of the lungs.

(a) *As regards its frequency.*

Hæmorrhage occurs at some stage of the disease in considerably more than half the total number of cases. The more carefully we inquire into the matter the more frequent will this symptom prove to be. Often, no doubt, the amount of blood may be insignificant, and the fact of hæmoptysis is sometimes forgotten. Wilson Fox found hæmoptysis in 161 out of 289 cases.* Turban found hæmoptysis in 235 out of 408 cases.† Walshe puts the frequency of hæmoptysis at 80 per cent.

(b) *As regards the cases in which hæmoptysis occurs.*

Hæmoptysis is comparatively infrequent in children, and is less frequent under 20 years of age than over 20. It again becomes somewhat infrequent in later life.

* Wilson Fox: *Treatise on Diseases of the Lungs and Pleura*, p. 787.

† *Beiträge zur Kenntniss der Lungentuberkulose*, p. 110.

Sex has been supposed by some authorities to have much influence, and menstrual disturbances have been alleged to predispose to hæmoptysis. It is unlikely that these considerations possess much importance.

Hæmoptysis has been supposed to be more frequent in the case of patients who present a family history of phthisis, but this is doubtful.

As regards the type of patient, no rules can be laid down.

(c) As regards the stage of the disease.

Hæmoptysis may occur at any stage of phthisis. It is often alleged to be the first symptom, but if these cases are closely investigated it will almost invariably appear that slight symptoms preceded the hæmorrhage and were ignored. These symptoms are usually cough, some loss of flesh, languor, debility, slight chills. Walshe found hæmoptysis to be the first symptom in 36 per cent. of cases. Lænnec says, "Le premier symptôme inquiétant et propre à donner l'éveil sur la maladie chez la plupart des phthisiques est ordinairement une hémoptysie." * Both these statements require some qualification. My experience is more in accord with the statistics of Turban, who found hæmoptysis the initial symptom in 47 out of 408 cases.† I feel sure, however, that in some of these cases slight but significant symptoms had preceded the occurrence of hæmoptysis.

Hæmoptysis may occur at any time in the course of phthisis, and may be the final symptom.

(d) As regards the amount of blood lost.

This varies within the widest limits. There may be only a few spits of blood, or several pints may be lost. It

* Auscultation Médiate, tome ii. p. 117.

† *Loc. cit.* p. 92.

might have been expected that early hæmorrhages would usually be small, late hæmorrhages large. This is often the case, but quite often it is otherwise. The first hæmorrhage may be profuse, the latest may be scanty.

(e) As regards the pulmonary condition which gives rise to hæmoptysis.

A good deal of obscurity hangs over this subject. Hæmoptysis may be due to local hyperæmia of vessels, to rupture of capillaries, to ulceration of a vessel in the progress of softening and excavation, to rupture of a pulmonary aneurism. Very profuse bleedings are usually due to the last-mentioned cause. It is often impossible to determine which of the above conditions is present in any individual case.

(f) As regards the appearance of the blood, and its chemical and microscopical characters.

In a typical case of free pulmonary hæmorrhage the appearance of the blood is very characteristic. It is bright red in colour, frothy, and has a "churned-up" appearance. Departures from these typical characters are, however, quite common. The blood may be dark and clotted, not mixed with air, but rather mixed with saliva; it may be salmon-coloured. These characters depend upon the seat of the bleeding, its rapidity, and the length of time that the blood has lain in the air-passages. Blood may be effused into a cavity, remain there sufficiently long to become dark and clotted, and then be ejected in this state. Or blood may remain for a time in the bronchial tubes or in the larynx or pharynx, and become more or less intimately mixed with the secretions of those parts. We must, therefore, interpret the physical characters of the blood with caution. I have never seen the blood of hæmoptysis

assume the typical "tarry" appearance so common in gastric hæmorrhage, except in those cases where blood coming from the lungs had been first swallowed and then vomited. Dried nodules, composed of blood, are sometimes ejected. Blood from the lungs is alkaline, blood from the stomach is acid. The blood in phthisis sometimes contains tubercle bacilli—more often not.

(g) *As regards the existence of a type of "hæmorrhagic phthisis."*

The division of cases of phthisis into a hæmorrhagic and non-hæmorrhagic group would be an artificial one.

Hæmorrhage occurs in considerably more than one half of all cases of phthisis. It may be an early symptom and never recur (this is rare); it may appear at any time in the course of phthisis; it may be a prominent feature of the closing scene. It has no definite prognostic significance. It does not mark any special type of patient nor any definite variety of the disease. Hence, a division of cases into hæmorrhagic and non-hæmorrhagic is not instructive or practically useful.

The term "hæmorrhagic phthisis" is sometimes used in a sense which has a certain limited degree of utility. It is sometimes applied to those cases where the principal feature is the occurrence of profuse hæmorrhages at long intervals, physical signs being slight or absent, and the constitutional state good. This type of phthisis, already frequently alluded to, has certain characteristics, *e.g.* a relatively favourable prognosis, readiness to respond to the air of high altitudes; but there is an awkwardness in labelling these cases "hæmorrhagic phthisis," inasmuch as they form only a small proportion of the total number of cases in which hæmoptysis is found.

(h) *As to the dangers of hæmoptysis and its prognosis.*

The dangers of hæmoptysis are either (1) Immediate, or (2) Remote.

The immediate dangers are not usually urgent, except in cases of advanced disease and marked debility. Patients with early limited disease rarely die of hæmoptysis. Death from hæmoptysis may be due to shock, suffocation, cerebral anæmia, syncope, or exhaustion. Shock is generally nervous in character, and rarely fatal. Suffocation may ensue from blocking of the air passages with blood. This is probably one of the commoner causes of death from hæmoptysis, and should be always remembered, inasmuch as it requires prompt and energetic treatment on quite different lines from those generally suitable to hæmoptysis. Death from cerebral anæmia and from syncope is probably commoner than death from exhaustion. The remote dangers of hæmoptysis are chiefly the establishment of a septic broncho-pneumonia, with the development of fresh foci of tuberculization in the lungs, and the transformation of a chronic into an acute or sub-acute case.

There is no definite prognosis in hæmoptysis. The fact of hæmorrhage does not stamp the case as either better or worse than the average. Everything depends upon the amount of blood lost, the frequency with which the hæmorrhage recurs, the effect upon the patient, the state of the lungs after hæmorrhage has subsided.

We have already considered in its broad outlines the question of diagnosis in connection with hæmoptysis. We shall now proceed to consider the differential diagnosis in a little more detail.

The distinction of hæmoptysis from hæmatemesis is usually easy. In the latter condition the blood is usually dark (often "tarry") in appearance, it is acid in reaction,

often mixed with food, vomited rather than coughed up, the vomit has often the well-known "coffee ground" character, the hæmorrhage is often preceded by pain in the stomach, or other dyspeptic symptoms, the bleeding usually ceases at once, but may recur, the motions may be "tarry." There is no cough, no persistent spitting of blood, and no rise of temperature. There are no signs in the lungs, but probably pain and tenderness in the epigastrium.

In hæmoptysis, on the other hand, the blood is often bright red, alkaline in reaction, mixed with bronchial mucus or saliva, coughed up rather than vomited, preceded usually by cough and other pulmonary symptoms, does not cease immediately, is often followed by a rise of temperature. There is no history of painful dyspepsia, no pain or tenderness in the epigastrium, no "tarry" motions, while there may or may not be physical signs in the lungs.

The above characters of hæmoptysis simply tell us that the blood has come from the lungs. They do not determine the cause of the hæmorrhage. Tubercle bacilli are rarely found in the blood of hæmoptysis, and the determination of the cause of the hæmorrhage must be settled by an analysis of the history, symptoms, and physical signs.

Difficulties in the differential diagnosis of hæmoptysis and hæmatemesis may arise in two ways, viz.—

- (1) Blood from the lungs may be swallowed, undergo certain changes in the stomach, and then be vomited.
- (2) Blood from the stomach may find its way into the lungs.

The history of the case will usually throw light on these points.

The distinction of genuine hæmoptysis from hæmor-

rhage having its seat in the upper air passages—bronchi, larynx, or pharynx—may be difficult. Something has already been said on this subject. In bleeding from the upper air passages the hæmorrhage is rarely profuse, often the sputum is only “streaked” with blood, it is mixed with saliva or bronchial mucus, in some cases the seat of the hæmorrhage can be seen. Fallacy may creep in from the circumstance that blood from the mouth, nose, or pharynx may flow into the lungs during sleep. In all cases the gums and naso-pharynx should be carefully examined. Hysterical girls, intent on exciting sympathy, often suck their gums and thus seek to add to the dignity of their sufferings by a specious show of hæmoptysis. Genuine hæmoptysis, depending on vaso-motor causes, seems to occur occasionally in hysteria.

Hæmoptysis from cardiac (usually mitral) disease does not usually cause much difficulty in the differential diagnosis. It is not uncommon, though rarer, I think, than might have been expected. It is commoner in mitral than in aortic disease, commoner in stenosis than in regurgitation, though not rare in this latter condition. It is generally found in cardiac cases where the other symptoms are prominent, but I have known hæmoptysis to recur persistently over a long series of years in a case of combined aortic and mitral disease, the patient meanwhile remaining fairly well, continuing at work, and having hardly any other symptom, except occasional attacks of vertigo. Such cases are rare.

Hæmoptysis from cardiac cause is not often profuse, and is not usually attended by much shock. Indeed, not uncommonly the patient professes himself relieved by the bleeding.

Hæmoptysis from certain blood dyscrasias—*e.g.* leucocythæmia, purpura, scurvy, hæmophilia, malignant infective

fevers—is rarely profuse, and the causal condition is usually obvious.

Hæmoptysis in my experience is rare both in chlorosis and in pernicious anæmia.

Among the rarer causes of hæmoptysis may be enumerated the following :—

Aortic aneurism.

Asthma.

Pulmonary infarction.

Pulmonary gangrene.

Emphysema.

Plastic bronchitis.

Bronchiectasis.

Hydatids of the lungs.

Carcinoma of the lungs.

Actinomyces of the lungs.

Chronic renal disease.

The *distomum pulmonale*.

In most of the above cases hæmoptysis is a subordinate feature. The history and the other symptoms will usually give the correct clue.

In some of these conditions the fact of hæmoptysis may give us a valuable hint for diagnosis, *e.g.* in aortic aneurism, carcinoma of the lungs, pulmonary infarction.

Profuse hæmorrhage, the result of rupture of an aneurismal sac, is usually obvious, and need not detain us. More important, from our present point of view, are those cases of repeated small hæmorrhages, believed to be due to “weeping of the sac” into the trachea or one of the large bronchi. These cases may simulate phthisis, and if it happen that the aneurismal sac compresses the lung, the simulation may be close. Often, however, the problem of differential diagnosis is not difficult. The subjects of aortic aneurism are commonly middle-aged men of robust

physique, and there is a history of syphilis, alcoholism, or strain; deep-seated thoracic pain and breathlessness are usually present, and the patient's arteries are atheromatous. The clinical picture is thus in most respects quite different from that of incipient phthisis.

I have met with hæmoptysis in quite a considerable number of cases of asthma. Perhaps such cases make an exceptional impression in the mind, inasmuch as they are unexpected. We have to inquire—

- (1) Whether the hæmorrhage is due simply to violent coughing. This I believe to be quite rare.
- (2) Whether ulceration of the bronchi is present.
- (3) Whether there is a quiescent tubercular lesion at one apex, accompanied by bronchiectasis.

An examination of the sputum may throw light on the case.

Pulmonary infarction is an important, though rare, cause of hæmoptysis. The presence of cardiac disease, the sudden onset, and the detection of a wedge-shaped area of consolidation in one of the lungs, will serve to clear up these cases.

Hæmoptysis may be a feature of emphysema, where it is probably due to degeneration of the pulmonary vessels; of plastic bronchitis, where an examination of the sputum will usually obviate any difficulty; of pulmonary gangrene, where the history and factor make diagnosis easy; of hydatids, actinomyces, or the presence of the *distomum pulmonale*, where in most cases a diagnosis is only possible by detection of the special parasite present. These cases do not call for any special comment.

A few words may be said regarding the remaining cases, viz. bronchiectasis, carcinoma, and chronic renal disease.

As a rule, bronchiectasis is easily distinguished from

phthisis by the chronic history, the fair constitutional state in spite of active pulmonary symptoms, the large amount of ill-smelling sputum, expelled at long intervals, the basic physical signs. If, however, hæmoptysis be a prominent feature, and the general condition bad, the simulation of phthisis may arise. The absence of tubercle bacilli from the sputum may be allowed great weight. The history, temperature, and situation of the physical signs will generally determine the diagnosis.

I reserve the case of carcinoma of the lungs for consideration hereafter.*

Hæmoptysis is much less common in chronic renal disease than might have been expected. It is rarely a source of danger.

Prognosis of hæmoptysis.

The prognosis of hæmoptysis depending on phthisis has been already considered.

In cardiac disease, hæmoptysis usually points to serious pulmonary congestion, and hence stamps the case as severe. The immediate danger is, however, for the most part slight. Often the bleeding relieves the patient's symptoms. Rarely does it precipitate a fatal issue. In pneumonia hæmoptysis has no prognostic significance. It does not portend special danger. It does not suggest tuberculosis. In the other conditions which have been enumerated, the prognosis depends upon the amount of blood lost, the persistency of the attacks, the degree of shock, the after condition of the patient. In the absence of severe shock, suffocative symptoms, marked syncope or much cerebral anæmia, the immediate outlook is usually favourable, and we shall often be justified in confidently assuring the patient that the immediate danger is slight.

* See Lecture XII., Some of the Rarer Forms of Pulmonary Disease.

In a certain proportion of cases hæmoptysis is followed by an infective broncho-pneumonia. This cannot be foreseen, so while the immediate outlook in hæmoptysis is so far as it relates to danger to life is commonly favourable, the more distant outlook is always doubtful.

Patients are very apt to overrate the importance of hæmoptysis, and to magnify its dangers.

Treatment of Hæmoptysis.

I shall first deal in some detail with the treatment of hæmoptysis depending on pulmonary tuberculosis. The other cases can then be dealt with somewhat summarily.

In an ordinary case of slight or moderate hæmoptysis occurring at an early stage of phthisis, active treatment is unnecessary, and usually does harm. The routine administration of internal astringents is a most pernicious practice. In the great majority of such cases the bleeding tends to stop spontaneously, and our task is simply to place the patient under the most favourable conditions for its arrest, and to endeavour to prevent its recurrence.

The patient should be put to bed in a well-aired room, the bed-clothes should be light but sufficient for comfortable warmth, absolute quiet and abstinence from conversation should be enforced, and little or no food should be given for several hours. A hypodermic of morphia and a saline aperient should be administered. In many cases no further measures are necessary. Our chief task at this stage is often to re-assure the patient, and to prevent panic and fussy interference on the part of friends.

It should be remembered that a certain degree of faintness is favourable for the arrest of hæmorrhage, and so long as the pulse continues good, this should not be interfered with. Sometimes, however, the case takes a less

favourable course. The hæmorrhage persists, and the amount of blood lost may give rise to concern. We have then to consider what further measures are necessary. The best remedy at this stage is opium, which may be pushed freely, if necessary. Patients suffering from hæmorrhage bear opium particularly well, and half a grain to one grain, may be given safely every two or three hours, if the bleeding is severe and persistent. Next to opium, I should place turpentine, which seems to me a remedy of some potency. I have seen nothing to give me confidence in the other internal astringents commonly employed—viz. tannic acid, gallic acid, dilute sulphuric acid, acetate of lead, ergot, chloride of calcium. Adrenalin is deserving of a trial, but so far there does not seem to be any decisive evidence of its value in pulmonary hæmorrhage.

The utility of astringent inhalations is doubtful. Inhalations of turpentine vapour are recommended on high authority, but upon the whole I think it is more certain that they excite cough—which we in such cases particularly desire to obviate—than that they exercise any astringent influence.

As regards the application of ice to the chest, I have only used it in a limited number of cases. I can recollect one case of persistent hæmoptysis, which resisted all the ordinary remedies, but ceased promptly on the application of ice. The fear that we may apply the ice over a spot other than the bleeding place need not concern us, as no doubt it acts reflexly upon the superficial nerves of the chest, or by quieting the heart. Care should be taken not to apply it over too large an area of the chest, or for too long a period.

An extreme degree of syncope or cerebral anæmia may compel us to resort to various methods of stimulation. These should be avoided, unless the urgency is great.

It must be insisted that in cases of moderate hæmoptysis in early phthisis the free exhibition of internal astringents is thoroughly bad practice. They lock up the secretions and aggravate the after effects of the hæmorrhage. Some diastrous cases have come under my notice where the reckless administration of these remedies has done much harm. We should bear in mind that while the patient thinks most of his hæmorrhage, we should think most of his condition after the hæmorrhage.

What are we to do when the hæmorrhage is alarmingly profuse or persistent? Much depends upon whether we have reason to believe that the disease in the lungs is early and limited in extent, or that there is extensive excavation. In the former case the prospects of the speedy cessation of the hæmorrhage are good, and the treatment, already outlined, may be adopted, perhaps somewhat more actively, but essentially on the same lines. If profuse hæmorrhage occurs in connection with extensive excavation of the lungs the danger is great. Nor is it certain that we can do much to avert it. In such cases, rupture of an aneurism in the wall of a cavity has probably occurred, and this accident is but little amenable to treatment. Opium is the best drug, and should be given with a generous hand. Nauseating doses of ipecacuanha, recommended by Trousseau; aconite, praised by Andrew; the artificial induction of pneumothorax, advised by Cayley; venesection, suggested by many authorities, may be enumerated as possible resources in these dire extremities. I have no experience of these methods. Slow-acting astringents are obviously futile. It must not be forgotten that the patient's most urgent danger may be death from asphyxia, owing to the blocking of the air passages with blood. In such cases all other considerations must yield to the urgent necessity of averting the imminent danger of

death. Active stimulation must be employed. As my former teacher, Dr. Sutton, of the London Hospital, was in the habit of saying, "Tell the patient to cough it up." Transfusion may sometimes be necessary.

The after treatment of hæmoptysis in phthisis is highly important, and is, perhaps, not sufficiently regarded. We find several types of case :—

- (a) The patient may profess himself relieved by the hæmoptysis. The sense of "tightness" in the chest is lessened, and the breathing is easier. These cases do not call for comment.
- (b) The hæmorrhage may subside in a few hours or in a day or two, and leave the patient's condition unaltered. In such cases rest and light diet should be advised for a few days, and the use of cod-liver oil or other nutrients and tonics should be temporarily suspended.
- (c) The hæmorrhage may be followed by broncho-pneumonia, usually septic in character. The temperature rises, new physical signs appear in the lungs, the clinical condition is definitely worsened. These cases call for much watchfulness. Rest, light food, purgation, a mild antiphlogistic course are indicated. A good combination in these cases is an effervescing saline mixture, with a few drops of morphia to each dose.
- (d) The hæmorrhage may be followed by the development of fresh foci of tuberculization in the lungs. The case passes from a chronic into a subacute condition. Rest, fresh air, a fairly generous diet, cold or tepid sponging, and a carefully regulated hygiene are now the chief desiderata.

The treatment of hæmoptysis from other causes than

phthisis need not detain us. In cardiac cases rest and purgation are the chief points. The use of digitalis need not be intermitted. Astringents are hurtful. Hæmoptysis depending on blood conditions seldom requires active interference. If treatment seems called for, it may pursue the lines suitable for moderate hæmorrhage in early phthisis. Hæmoptysis is rarely the leading feature of hæmophilia. Where it requires treatment in this condition, turpentine and perchloride of iron are the best remedies. In pneumonia, hæmoptysis does not call for any special treatment.

LECTURE XII.

SOME OF THE RARER FORMS OF PULMONARY DISEASE.

SUMMARY.

Malignant Disease of the Lungs.

Clinical details regarding three cases.

Physical signs of malignant disease, their diversity and misleading character.

Symptoms and history.

Presence of malignant disease in other organs.

Duration.

Atypical Pneumonia.

Latency of physical signs, abnormal course of the temperature, delayed crisis, absence of pyrexia.

The so-called "gastric" pneumonia.

The "hepatic" type.

The "cerebral" type.

Question of pleuro-pneumonia.

"Larval" pneumonia.

Pneumonia in old age.

Cases of "spreading congestion."

Foreign Body in the Bronchi.

History of case.

Malignant Disease of the Lungs.

Malignant disease of the lungs is sufficiently rare to lend an interest to any case of the kind. I have met with three cases in my practice. In only one of the three was there the opportunity of verifying the diagnosis at autopsy, but in the remaining two the history of antecedent malignancy in other organs, the nature of the symptoms, the course

and event of the cases made the diagnosis practically certain. I will give the details of the three cases.

CASE I.—The patient was a woman, aged 46. The symptoms in this case were very confused, inasmuch as hectic fever was a pronounced feature, and at the autopsy pus was found in the affected lung, which also contained a large mass of encephaloid cancer. The duration of the case was a little over four months. Apart from the signs in the lungs, the only clue to diagnosis at an early stage was afforded by a firm nodule, an inch and a half in diameter, which was adherent to the skin of the left shoulder.

The physical signs were as follows: Over a large portion of the right lung, in the median and basic regions posteriorly, there was dulness, diminished tactile fremitus, and absence of breath sounds. A few rhonchi were audible in various parts of the chest. The heart was not displaced. There was much pain in the chest, with a good deal of dyspnoea. Expectoration was abundant, but did not contain either blood or tubercle bacilli. At the autopsy it was found that a large mass of encephaloid cancer occupied the root of the right lung, and compressed the right lower lobe, which was collapsed and splenized. Three additional nodules, two upon the abdominal wall, and the third upon the left thigh, appeared during the course of the case, and led to a suspicion of its true nature.

The physical signs in this case were for a time undistinguishable from those usually present in pleural effusion.

CASE II.—The patient was a young gentleman, aged 21. In the year 1894 his left leg had been amputated for osteo-sarcoma at the knee-joint. In the spring of 1899 the patient began to spit blood and to show signs of failing health. There was fever, and some night-sweating, and

physical signs manifested themselves in the right lung. Hæmorrhage was for many weeks persistent and uncontrollable. There were no tubercle bacilli in the sputum. Pain in the chest was a prominent feature.

The physical signs consisted of patches of dulness and crepitation in the right lung, in its upper and median portions posteriorly, the apex not being involved. For a time the exclusion of tuberculosis was a difficult matter. The history, symptoms, and physical signs would have been consistent with that diagnosis ; but, on the other hand, there were no tubercle bacilli in the sputum, the right apex did not share in the morbid process which was going on, pain in the chest was a prominent and persistent feature, and there was the history of antecedent osteo-sarcoma. On these grounds, it was thought probable that there was a malignant deposit in the right lung.

The progress of the case left no room for doubt as to its nature. The right lung became solidified, and pushed the liver downwards. The left lung became involved, the pyrexia and hæmoptysis ceased, but emaciation continued to progress. The patient died upon October 19, 1899, the duration of the case from the first onset of definite pulmonary symptoms having been about eight months. This patient was rheumatic, and had well-marked joint-affection, which may have had some relation to the pyrexia.

This case was interesting chiefly because the simulation of phthisis was close. Hæmoptysis, pyrexia, night-sweating, and progressive emaciation, with the presence in the right lung of well-marked physical signs not at first distinguishable from those of pulmonary tuberculosis gave rise to difficulty. The points which were chiefly relied upon in the diagnosis were as follows :—

(a) The absence of tubercle bacilli in the sputum,

This fact does not, of course, exclude tuberculosis, as it is well known that cases of phthisis may run their course without bacilli ever being discovered in the sputum. The explanation of this phenomenon is a pathological question, but the clinical fact is certain. In the present case, however, there was active disease, well-marked and progressive physical signs, and abundant expectoration. With such conditions, the absence of bacilli was of course very significant.

(*b*) The non-implication of the apex. The cases of phthisis where a lesion of considerable extent and progressive character does not involve the apex must be very few.

(*c*) The prominence and persistent character of the thoracic pain.

(*d*) The persistency of the hæmoptysis. This continued for months almost without intermission, in spite of every form of treatment. This is, in my experience, very rare in phthisis.

Finally, the displacement of the liver and the pressure symptoms made the case clear.

The symptoms, as a whole, were not distinguishable from those of phthisis.

CASE III.—The patient was a lady, aged 61.

The right mamma was excised for scirrhus on June 2, 1900. The patient made a good recovery from the operation, and continued well for six weeks. She then began to complain of shortness of breath, and of pain in the right chest. At this time the scar and the skin in its neighbourhood were quite healthy, and there was no ulceration or induration. The pulse and temperature were normal. There were no physical signs in the lungs.

Towards the end of August the breathlessness and the pain in the chest increased. There was a slight cough with frothy expectoration, no hæmoptysis, morning temperature normal, evening temperature about 100° . The physical signs at this stage were those of general bronchial catarrh. In a few days, however, dulness with impaired vesicular breathing appeared on the right side in front. There was now some irritation in the scar. Dyspnœa, cough, and expectoration rapidly increased, and the sputum became tinged with blood. The area of dulness and diminished breath sounds went on increasing, and extended upwards. In the last week of her life the patient suffered from severe dyspnœa, cyanosis, frequent cough, and hæmoptysis. The temperature continued slightly febrile in the evenings. There was slight œdema of the neck and right upper extremity, and some enlargement of the cervical and the axillary glands on the right side. The patient died upon September 16, 1900, fifteen weeks after the operation for scirrhus of the breast, and nine weeks after the first appearance of pulmonary symptoms. No autopsy was obtained.

These cases, too few in number to justify any dogmatic conclusions, suggest the following observations:—

- (a) In malignant disease of the lungs, the physical signs present great diversity, and are not pathognomonic. In Case I. the physical signs resembled those of pleural effusion; in Case II. they simulated those of phthisis; in Case III. they were at first simply those of general bronchial catarrh, though after a short time they became specialized and significant.

If the physical signs in pulmonary malignancy are at first obscure, they tend to become clearer as time

goes on. An area of dulness and diminished breathing tends to develop, and this area is neither, as a rule, apical nor basic, but in the general substance of the lung or at the root. It must be borne in mind that the physical signs are due in part to the new growth directly, in part to its pressure effects upon the surrounding lung, and in part, sometimes, to co-existing pleural effusion. We may thus have a somewhat confusing clinical picture.

In none of my cases was well-marked bronchial breathing present. I think this may be explained as follows: Over the area of the malignant growth the consolidation is very dense, and involves the bronchial tubes. Over the collapsed and splenized area in the neighbourhood of the growth the tubes do not remain patent. Under such conditions the breathing will be weak or absent, rather than bronchial.

- (b) The symptoms most characteristic of pulmonary malignancy are pain, usually prominent and persistent, dyspnœa, and blood-stained expectoration. In one of my cases hæmoptysis was absent. Pyrexia, night-sweating, and rapid emaciation may all be present.
- (c) The history will often supply the necessary clue. Malignant disease of the lungs is almost always secondary to malignant disease elsewhere in the body.
- (d) A careful search should be made for malignant disease elsewhere. In many cases malignant disease of bones, mamma, uterus, or other organs will be evident. Sometimes the primary seat of disease may be a small nodule (as in Case I. of my series), which may escape attention.
- (e) The duration of these cases is short—much shorter than the average duration in cases of phthisis.

Walshe gives an average duration in pulmonary malignancy of 13·2 months. My cases would suggest that 6 months would be more correct.

Atypical Pneumonia.

(a) Latency of the physical signs.

I can recall a considerable number of cases where the physical signs were for several days entirely latent, although the history and symptoms left no room for doubt as to the true nature of the case.

One case was much impressed upon me at an early stage of my practice. The patient was seen, with me, by the late Dr. James Smith and Dr. (now Sir) W. Whitla. She was a little girl, aged 4, who took suddenly ill with severe pyrexia (104°), marked dyspnœa, herpes about the mouth, pain in the chest. The symptom-complex was typical of pneumonia. But for several days no physical signs whatever could be discovered, although diligent search was made for them, and the diagnosis seemed certain. There was no sputum, as might be expected in so young a child. On the fourth day typical dulness and tubular breathing, and the case ran the usual course of pneumonia, terminating favourably.

Stokes believed that before the advent of the so-called "primary crepitus," the vesicular murmur became harsh or rough. We rarely have the opportunity of examining patients at this stage. Weakening of the breath sounds has, in my experience, often preceded the development of bronchial or tubular breathing.

(b) Abnormal course of the temperature.

Of the normal course of the temperature in pneumonia I need say nothing. The sudden rise, the high fastigium, the crisis occurring from the fourth to the tenth day, the rarity of any secondary rise are well known. Departures

from the typical course are, however, common, and must be cautiously interpreted. If we shake our heads over every irregular chart in pneumonia, we shall sometimes excite needless fears.

I exclude from consideration cases of "pneumonic phthisis," which may generally be recognized by care as regards history, and a thorough examination of the sputum.

I have seen no case of a gradual rise of temperature in pneumonia, but in a few cases the rise may be by two or more rapid increments, *e.g.* a temperature of 100° on the first night may be followed by a temperature of 103° or 104° on the third or fourth day. These cases are not uncommon, and may be somewhat misleading. Their most probable explanation is that the first rise of temperature is coincident with the development of a small pneumonic patch in the lung (which may subside at this point), and that the higher temperatures mark the rapid invasion of larger tracts of lung.

Delayed crisis is not uncommon, and may have several explanations. It may be due to *extension of the disease overlapping the primary affection*, as in the following case :—

G. H., a boy of 6, took ill upon December 20, 1900, with cough and feverish symptoms. On December 22 signs of left basal pneumonia were discovered. For several days the patient's condition remained without material change, the general symptoms being severe. On January 1, 1901, signs of pneumonia of the right apex appeared. No change of importance took place until January 7–8 (the twentieth day of the disease), when the crisis occurred, the temperature falling from 103° at night to 99° the next morning. Convalescence was, thenceforward, rapid and uninterrupted. It seems clear that the long delay of the

crisis in this case was due to secondary implication of the right apex overlapping the primary affection of the left base.

Delayed crisis may be due to *the presence of complications*. Of these empyema is one of the most important. Of this complication I have seen many examples. Where as a pneumonia wanes signs of effusion in the pleural cavity appear, we should always suspect the presence of pus. General bronchitis is a much rarer complication. I have met with some examples, and I regard this complication as a grave one.

Delayed crisis may be due to *the influence of age*. It is common in the elderly, and in such cases it has only a minor degree of significance. Pneumonia is always a grave condition in the elderly (after the age of sixty rather more than half the cases die), but a delayed crisis and a somewhat tardy convalescence may be regarded in such cases as more or less normal events, and do not possess much prognostic importance.

In rare cases the pneumonic pyrexia subsides by lysis and not by crisis.

As a rule, when the crisis is accomplished (usually in from twelve to thirty-six hours), there is no further rise of temperature, but a slight rise for one or two evenings is not very uncommon, and has no special significance.

Pneumonia, as is well known, may run its course either absolutely without fever or with only a brief and trivial rise of temperature. I remember a case in my experience of a young gentleman, aged 35, who had been of very intemperate habits, and who died of an attack of pneumonia, with typical physical signs, death occurring on the third day. There was no rise of temperature until a few hours before death.

(c) The presence of misleading symptoms.

We may recognize a "gastric" type of pneumonia, an "hepatic" type, and a "cerebral" type. These terms are not free from objection, but they may serve the useful purpose of reminding us that pneumonia, usually one of the most defined and characteristic of diseases, is somewhat protean in its characters, and may assume unwonted forms.

In the "gastric" type the symptoms are gastro-intestinal in their more prominent features. These cases have been sometimes mistaken for appendicitis, or perforating gastric ulcer. In the "hepatic" form jaundice and pain over the liver are prominent features. A diagnosis of "congestion of the liver," or of gallstones, has sometimes been given in these cases. Attention to the temperature, the breathing, and the physical signs in the chest will usually obviate any difficulty in diagnosis.

The "cerebral" type is quite common, and is the cause of many unfortunate errors of diagnosis. Violent delirium and headache—in children, convulsions—are prominent features. A diagnosis of meningitis has often been given in these cases. This error can usually be avoided by attention to the breathing, the movements of the *alæ nasi*, and the physical signs in the chest.

In all cases of sudden febrile illness with marked symptoms, the possibility of pneumonia should be borne in mind, and the occasional latency of the physical signs should not be forgotten. In a child, a sudden illness attended by a temperature of 103° or upwards, quick breathing and marked movement of the *alæ nasi*, should suggest the probability of either capillary bronchitis or pneumonia.

Profuse hæmoptysis sometimes occurs in pneumonia, and may excite unfounded fears of tuberculosis. Its

cause is not quite clear, but its prognostic importance is small.

Genuine pleuro-pneumonia is not common in the human subject. Of course, a certain amount of pleurisy accompanies every case of pneumonia, but this is usually confined to the early stage of the attack, and is soon lost in the pneumonic symptoms. Yet genuine pleuro-pneumonia sometimes occurs—*i.e.* a case where pleural signs persist throughout the pneumonic attack and play an important part in determining the clinical condition. It is well to take a guarded attitude towards such cases. My experience of them is that they are usually slow in their course, that empyema sometimes develops, but that in other cases recovery, though somewhat tardy, is complete and satisfactory. The suspicion of tubercle may arise, but it is well to reserve our opinion on this point until we have had time to watch the progress of the case and to examine the sputum.

In some cases it is not easy at the outset to decide whether a case with a complex clinical picture is going to turn out pleurisy or pneumonia. Pleurisy may have a "pneumonic" onset, pneumonia may be accompanied by pleurisy, and the special pneumonic signs may be for a time latent. In such cases, of which I have seen several examples, I believe our best guides are the state of the breathing and the amount of physical prostration. Sputum may be absent, and the range of the temperature will not always settle the question at issue. But the dyspnœa of pneumonia is much more of a genuine "air-hunger" than the painful and halting, rather than urgently breathless, respiration of pleurisy. The pneumonic patient is also, for the most part, much more prostrate than the sufferer from pleurisy. Often he lies like a log in bed, and turns with difficulty for the purpose of physical

examination. We do not observe this degree of prostration in pleurisy. If any expectoration be present, its nature may at once put an end to any doubt as to the nature of the case. As Aufrecht remarks, "One single pneumonic sputum confirms the diagnosis." *

A type of pneumonia has been described under the title of "larval," "masked," or "ephemeral" pneumonia. The patient presents the initial symptoms of pneumonia but in moderate degree. Slight implication of the lung can be recognized, but the attack aborts in forty-eight hours or less, and the patient rapidly convalesces. Sir J. W. Moore has described an interesting case of this kind, where there could be no doubt of the diagnosis.† I have often suspected this condition at the bedside, but from the nature of the case a positive diagnosis is rarely possible. Some of the cases of "smart feverish colds," with pain in the chest, some degree of dyspnœa and labial herpes, probably belong to this category. If a diagnosis is not at first always possible, we are not left long in doubt. Should the case be one of "larval" or "ephemeral" pneumonia, a rapid subsidence of all symptoms in two or three days may be expected.

Pneumonia in old age may present considerable diagnostic difficulties. The onset is not always well marked, the symptoms are somewhat indefinite, the temperature is moderate or low, the sputum does not possess the typical "rusty" character, the physical signs in the chest may be ill-defined. It is highly important to recognize the disease, as it is a very deadly one in such cases, and prompt treatment is imperative. Pneumonia in the elderly often occurs as the terminal event in some

* Nothnagel's *Encyclopædia of Practical Medicine*, Art. Inflammation of the Lungs, E. Aufrecht, M.D.

† *Encyclopædia Medica*, Art. Pneumonia, vol. v., p. 436.

chronic and exhausting disease, *e.g.* cancer, chronic nephritis, disease of brain or cord. A careful examination of the chest should be practised in such cases, whether there are any obvious pulmonary symptoms or not.

I have seen several examples of a condition—no doubt familiar to most clinicians—which may or may not be an atypical form of pneumonia. The patient takes suddenly ill with cough, oppression of breathing, and prostration. He becomes more or less cyanosed, the pulse is very frequent and of low tension, the extremities are cold, the skin is bathed in perspiration. On examination, fine, moist, crepitant râles are heard over a considerable area of one lung. There is no appreciable dulness, and no bronchial breathing. The area of crepitation gradually becomes more extensive. The temperature is only slightly raised. Whether this form of “spreading congestion” is a variety of croupous pneumonia or not is a question. Heart failure and pulmonary œdema seem to play a considerable part in these cases.

Most of the cases of this kind which I have seen have terminated in death, usually within two or three days.

Foreign Body in a Bronchus.

With the ordinary features of foreign body in a bronchus we are all familiar.

The following case shows that difficulties may arise in connection with this condition :—

A.B., a gentleman of about forty years of age, consulted me in April, 1899, for a hard, irritating, violent and spasmodic cough, which had lasted for six or seven months, and had resisted every method of treatment. In the previous September the patient had swallowed a fish-bone, which “had gone the wrong way.” In January, 1899,

he had an attack of pneumonia of the left apex, and had convalesced in about a month.

At the date of my examination—April, 1899—the patient was found to be in robust general health, weight 14 st. 4½ lbs., temperature normal, pulse 66. The only complaint was of violent and incessant cough. On examination, there was tenderness on pressure over the third left costal cartilage, and under the left clavicle the respiratory sounds were a little indistinct. There was no expectoration.

The questions at issue were—

- (a) Did the whole case turn upon the lodgment of a fish-bone in the left bronchus, and was the left apical pneumonia connected with its lodgment there; or
- (b) Was the history misleading, and was the real condition either tuberculosis of the left apex, or commencing aneurism of the aorta?

No confirmatory evidence either of tubercle or of aneurism could be discovered.

Some months afterwards the patient coughed up a fish-bone, and was at once relieved of his troubles.

LECTURE XIII.

SOME THERAPEUTIC PROBLEMS IN PULMONARY DISEASE.

SUMMARY.

A. Treatment of Pneumonia.

Ancient and modern methods, heart failure the chief source of danger.

Indications—

(a) To reduce the strain upon the heart.

(b) To sustain the strength of the heart.

(c) To abstain from depressing the heart.

Importance of rest, good nursing, suitable food. Dangers of over-feeding.

External applications—poultices, ice, sponging.

Question of venesection.

Use of morphia, alcohol, strychnia, digitalis, quinine, oxygen.

Indications and contra-indications as regards alcohol.

Question of the value of the cardiac tonics.

Objections to remedies of the aconite class.

B. Treatment of Empyema and its Complications.

Broncho-pleural fistula, arguments for and against operative measures, question of "spontaneous cure" in these cases.

C. Treatment of Asthma.¹

Question of the seat of irritation—(a) in bronchial tubes, (b) in digestive tract, (c) in naso-pharynx, (d) in other localities.

Use of iodide of potash, arsenic, nux vomica, lobelia, caffein, grindelia.

Symptomatic treatment.

Use of remedies of the stramonium class.

Influence of locality and climate.

A. Treatment of Pneumonia.

The treatment of pneumonia has been one of the great battle-grounds of medicine. One must speak with bated breath on a subject so controverted, so much

associated with erroneous practice in the past. There are those who look back regretfully to the days when venesection was the routine practice in pneumonia, and who believe that we have surrendered a valuable therapeutic method. I do not share these regrets. I have seen venesection employed in a few cases of pneumonia, and I do not desire to see it again accepted as a routine method of treatment. Its dangers are manifold ; its advantages are problematical ; it is open to great abuse. That venesection sometimes mitigates symptoms and relieves a labouring right ventricle is, of course, certain, but that it does any real good in the long run in pneumonia seems to me doubtful. The pendulum has however, swung far in the opposite direction. A crass method of over-feeding and indiscriminating stimulation has been too often adopted, without regard to the patient's peculiarities or the special indications of his case. This method, also, has its dangers—less grave than those of routine venesection, but not unimportant.

We do not yet know the pathology of pneumonia. That it depends upon the action of micro-organisms is highly probable, but the proof that the pneumococcus is the sole cause is still far from convincing. We may hope some day to have a specific remedy, something in the nature of an antitoxin. In the mean time, the therapeutic problem which confronts us in pneumonia may be stated somewhat as follows: The patient is the subject of an acute specific febrile disease, which throws a special strain upon the heart, usually self-limited, rarely continuing beyond the ninth or the tenth day. The morbid-process in the lung tends irresistibly towards spontaneous cure, if the patient survives long enough. The therapeutic indication is to keep the patient alive until the disease has exhausted itself. We can do little or nothing to shorten

the attack or abate its severity. We can do much to assist the patient to withstand it successfully.

The special danger in pneumonia is heart failure. "Patients who die of pneumonia are killed by cardiac insufficiency," says Von Jürgensen. Perhaps this view is somewhat exclusive, but it is, in the main, sound. I have, however, seen a patient gradually sink from pneumonia, the pulse keeping about 80, regular and of good tension, until near the end. Œdema of the lungs is usually a prominent feature in fatal cases. Sudden death is comparatively common. The patient does not, at least in the great majority of cases, die of dyspnœa or asphyxia. He dies, in most cases, because his heart succumbs to the strain upon it.

What is the determining cause of heart failure in pneumonia? Not myocarditis, which is not usually marked in fatal cases; not the severity of the fever, for in some of the worst cases the temperature is at no time high. Probably toxæmia is the usual explanation.

Two facts, then, stand out strongly: The patient is engaged in a struggle, usually sharp, always short, and his chief danger is heart failure.

The therapeutic indications are, then, clear, viz.—

- (a) To reduce the strain upon the heart.
- (b) To sustain the strength of the heart.
- (c) To avoid any method of treatment which, however palliative to other symptoms, might tend to wear out the strength of the heart.

Under the first head we may include perfect rest, the relief of pain, the promotion of sleep. The patient should be kept perfectly quiet in a well-aired room. He should not receive any visitors or engage in any general conversation. He should not leave his bed for the purpose of relieving the bladder or rectum. For the relief of pain

and the promotion of sleep, I believe the moderate use of morphia in the early days of pneumonia to be both safe and useful. At a later stage its use requires to be very guarded. A poultice, light, warm, and frequently renewed, seems generally welcome to the patient, and is apparently comforting. It should not be continued for more than two or three days. Ice to the chest has been recommended, but has not come into general use. We have to consider whether the application of ice might not in some cases tend to depress the heart. Tepid sponging, with the addition of eau de Cologne or other fragrant spirit to the water, seems to refresh the patient and relieve the nervous symptoms. Its effect upon the pyrexia is slight.

There seems good reason for not pushing antipyretic measures in pneumonia. The pyrexia, though often severe, is short, and has hardly time to produce much granular degeneration of the heart-muscle. Statistics show that cases with a somewhat sharp pyrexia often do better than those with a lower temperature, while afebrile cases are the most deadly of all. It is to be remembered that almost all antipyretic drugs are cardiac depressants, and hence likely to be hurtful in pneumonia.

The second indication is to sustain the strength of the heart. We seek to fulfil this indication by regulating the diet, by the use of alcohol, and by the administration of such remedies as strychnine, digitalis, caffeine, and quinine.

Much harm is, I believe, done by over-feeding in pneumonia. The patient's powers of digestion and absorption are almost in abeyance, and filling his stomach with undigested food can hardly be for his benefit. Further, an overloaded stomach will press against the diaphragm, and will further embarrass the labouring heart. I believe the amount of nourishment should be moderate,

it should be as nourishing and as easily assimilated as possible. Milk fulfils these indications best, and in a majority of cases is the only necessary food. The amount should not exceed three pints in twenty-four hours. If it seems desirable to supplement this diet, small quantities of fresh beef-juice or egg-flip may be given. A cup of tea is harmless and refreshing. All starchy foods should be withheld, as likely to create flatulence. Simple refrigerant drinks may be given moderately, care being taken not to distend the stomach. The lavish and indiscriminate administration of excessive quantities of nourishment may lead to acute dilatation of the stomach and precipitate a fatal issue.

The question of the value of alcohol in pneumonia is an important and much controverted one. Alcohol has been sometimes immoderately praised, sometimes condemned as useless or hurtful, sometimes recommended for occasional use according to the nature of the symptoms. Amongst those who, upon the whole, favour the use of alcohol in pneumonia, may be mentioned Germain Sée, Von Jürge nsen, Fagge, and Osler. Amongst those who, upon the whole, discourage its use may be enumerated Skoda, Hughes Bennett, Strümpell, Aufrecht, and J. W. Moore. Most observers are agreed that young patients generally do well without alcohol, and that old patients and the intemperate require it. But the question still remains open, Is alcohol to be recommended in the majority of cases of pneumonia or not? Has it any valid claim to be regarded as a routine remedy? To this question I am disposed to give a negative answer, while inclining to the moderate use of alcohol, where its use is indicated by the state of the pulse and of the nervous system. It may be accepted that in proportion as the symptoms assume the "adynamic" or "typhoid" type, the indication for alcohol

becomes more decided. With prostration, cyanosis, cold extremities, rapid feeble pulse, probably few would hesitate to give alcohol. But, in point of fact, these rules do not take us far. They cover cases where controversy is hardly likely to arise. My own practice is not to give alcohol if (a) the symptoms are "sthenic," (b) the pulse fairly good, (c) œdema of the lungs absent, (d) the nervous condition fairly good. As regards the form and amount of alcohol, I think brandy is upon the whole the best form, and that the amount should be from 3 to 4 ounces as a minimum, to 6 to 8 ounces as a maximum. Even so judicious a practitioner as the late Hilton Fagge advised heroic doses of alcohol—as much as 20 ounces in the day—in certain cases of pneumonia. I believe this to be bad practice. I have never seen any good from doses of such magnitude, and I am inclined to think they are usually hurtful.

While we may often justifiably hesitate whether to give alcohol or not in pneumonia, I believe we shall seldom have much doubt whether it is doing good or harm. In some cases the benefit from its administration is clear. The patient seems refreshed, his sufferings are diminished, the skin acts more freely, the nervous and the circulatory condition improves. Under such circumstances we feel confident that alcohol is doing good. But who has not seen the reverse picture? Who has not seen alcohol given *secundum artem*, although the patient protested that he felt uncomfortable after its use, that his skin felt hotter than ever, that his breathing was more embarrassed than before? We must not forget that the effect of alcohol upon the organism in health varies widely. To one person it is an almost pure stimulant; to another, an almost pure narcotic. I see no reason for believing that these personal idiosyncrasies disappear under the stress of

acute disease. It is just as reasonable to suppose that they may be intensified under such conditions. Therefore, caution seems advisable in the use of alcohol. In pneumonia, apart from obvious and incontestable indications, it should not be given as a routine measure, its effects should be carefully watched, and we should be ready to stay our hand if we have just cause to suspect that it is doing harm.

As regards the value of drugs in obviating heart failure, we find considerable divergence of view. The remedies usually recommended are the following: digitalis, strophanthus, caffein, strychnine, ammonia, ether, oxygen. I have made an extensive trial of digitalis in pneumonia, though never in the heroic doses recommended by Traube, Fickl, Petrescu, and others. I am inclined to think it possesses a certain value, although its utility is far less certain and manifest than it is in cardiac disease. G. W. Balfour recommends digitalis in drachm doses of the tincture in the precritical collapse of pneumonia.* We must remember that digitalis is somewhat slow acting, and that if we are to rely upon it at all in pneumonia we must give it in adequate and quickly repeated doses.

Strophanthus and caffein I have not largely tried. Strychnine I have used largely, and I have no doubt that it is often of great value. It should be given hypodermically, and in adequate doses (m 5-8 of the liquor) repeated as often as may seem advisable. The effect upon the pulse is sometimes at once manifest.

Ether and ammonia have only a very limited utility. Oxygen is a remedy about which there is much difference of opinion. After many trials, I have come to the conclusion that it possesses some value, especially when cyanosis is present. Practitioners who give oxygen only in severe

* The Senile Heart, p. 259.

cases and when patients are *in extremis*, will naturally form a very unfavourable opinion of its utility. It should not be reserved for such emergencies. Patients often profess relief from its use, and call for the remedy. It may be given at short intervals for five minutes at a time. It is sometimes advised that the gas should be warmed.

Quinine is warmly praised by some observers in pneumonia. How it acts, if its virtues are genuine, is a question—not by its antipyretic action, in all probability. Very large doses are not desirable.

Expectorants are often given in pneumonia, probably on the basis of a false analogy with bronchial affections. They are probably quite useless.

Our third rule is not to give remedies which, however they may relieve other symptoms, tend to weaken the heart in the struggle before it. Aconite is a case in point. All cardiac depressants should be strictly avoided in pneumonia.

The treatment of pneumonia remains a highly unsatisfactory problem. Our present methods must be regarded as provisional and inadequate. No doubt, some day we shall improve upon them. In the mean time it is well to avoid a return to the exploded methods of the past, and to employ our present resources in a critical spirit, distinguishing case from case, and endeavouring to profit by any special indications which may be present.

B. The Treatment of Empyema and its Complications.

The general principles of the treatment of empyema are well established.

When the presence of pus in the pleural cavity has been proved, the operation of incision (with or without the excision of one or more ribs) should be performed, and adequate drainage secured. Washing out of the pleural

cavity with antiseptic solutions is usually unnecessary and not free from risk. In most cases the putridity of the pleural contents is corrected by drainage alone.

Aspiration, without incision, probably cures a few cases, especially of pneumococcal empyema in children, but it cannot be relied upon, and cannot take the place of the larger operation. Aspiration as a preliminary to incision and drainage is often advisable.

Where empyema has been followed by the formation of a broncho-pleural fistula the mode of procedure is not so certain. In proportion as cases of empyema are early recognised and promptly submitted to operation this accident should become rare, but it still occasionally occurs. The question then arises whether with a free exit for the pus *per vias naturales* there is any advantage in incising the chest. It is well known that these cases in not a few instances undergo a spontaneous cure. Their after history is, however, not so well established. During the last few years I have had under observation three cases of broncho-pleural fistula following empyema, in which for various reasons no operation was performed. In one case the patient positively declined operation. In the remaining two the surgeon, after inserting the exploring syringe, could not satisfy himself that any considerable amount of pus remained for evacuation, and decided to proceed no further. All three cases made a good recovery at the time; one of the three, however, after a lapse of several years became phthisical.

The general opinion is in favour of operating in these cases. Rosenbach says, "If it can be positively determined that an empyema has ruptured through the bronchi, an empyema operation is to be advised, in spite of the fact that spontaneous cure in these cases not rarely occurs; because operation, provided the lung retains some power

of expanding, is certain to hasten recovery, and is often the only means of curing the patient. To depend on spontaneous cure in such cases is not rational, as the usual results of pleurotomy present better chances for recovery than the uncertainty of internal evacuation, which fails to guarantee thorough elimination of the pus and permanent asepis."*

It would be interesting if we had fuller details of these cases of "spontaneous cure" following broncho-pleural fistulæ, consequent on empyema. They are not uncommon, but their after history is not clear. Is the cure complete or incomplete? Do any considerable proportion of these cases ultimately become phthisical? Would that tendency have been obviated by timely operation? How far might a phthisical tendency in such cases be eliminated by hygienic treatment or a residence at high altitudes?

These are interesting and important questions, but they can hardly be confidently answered in the present state of our knowledge.

The balance of evidence is at present in favour of treating these cases on the same lines as empyema without fistula.

C. The Treatment of Asthma.

The treatment of asthma is a much controverted question. It is, as is well-known, one of the happy hunting-grounds of the quack. Nor is this surprising. The disease is eminently erratic, subject to psychical influences, affected in a most potent yet most mysterious manner by the influence of locality, responding now to one remedy and again to another, sometimes baffling all our therapeutic endeavours, sometimes improving under the most unlikely conditions. Such a disease lends itself to

* Nothnagel's *Encyclopædia of Practical Medicine*, Art. *Pneumothorax*.

irregular methods, and presents peculiar difficulties as regards sound and scientific treatment.

The term "asthma" is a clinical rather than a pathological one. By it we understand a form of intense recurrent dyspnœa, often markedly periodic, attended in many cases by a characteristic sputum, probably reflex in origin, not generally involving any serious danger to life, and usually accompanied by some degree of bronchial catarrh.

That asthma is a neurosis can hardly be doubted, and that it depends upon reflex irritation seems highly probable. The seat of irritation is in the great majority of cases in the respiratory passages, occasionally in the digestive tract, only very exceptionally elsewhere. Of asthma depending upon irritation in the urinary and generative organs I have no experience, but such cases are described. The question arises, In what part of the respiratory tract is the irritant to be found? Some authorities attach much importance to sources of irritation in the naso-pharynx as causes of asthma, and operative measures based upon this theory have often been undertaken. My experience of this doctrine and of the practice based upon it is unfavourable. That nasal obstruction, adenoid growths in the pharynx, and other such causes may produce forms of dyspnœa simulating asthma, and may admit of cure by operation, may be allowed. It is also true that the so-called "sensitive areas" are found in the naso-pharynx in a considerable number of cases of asthma. But I have seen nothing to convince me that asthma of the classical type is ever due to nasal or pharyngeal causes, or is ever cured by operation. I exclude from consideration the special case of hay-asthma, where the seat of the disease is no doubt nasal.

Nor does the seat of irritation in genuine asthma

appear to be often in the larynx. I have no experience of asthma supervening upon laryngitis, acute or chronic, or definitely affected by remedies directed to the larynx.

The seat of irritation would appear to be mainly in the bronchioles. Fraenkel is of opinion that there is always an epithelial inflammation of the mucous membrane of the bronchioles, in which a remarkably abundant epithelial desquamation takes place.* It is, of course, a question whether the nervous element in asthma—that there is a nervous element in the disease hardly requires demonstration—is primary or secondary, *i.e.* whether inflammation of the bronchioles is the primary fact setting up an irritation of the nervous system, or whether a disturbance of the nerve-centres sets up a vaso-motor turgescence in the bronchioles. The history of these cases would suggest that the former alternative is much the more usual.

I do not propose to enter more fully into the question of the pathology of asthma, as my immediate purpose is the consideration of the therapeutic problem.

Before entering upon the treatment of a case of asthma it is well to make sure that none of the following conditions are present, *viz.* heart disease, renal disease, tuberculosis of the lungs, adenoids, nasal obstruction. It is obvious that any of these conditions, if present, will alter the therapeutic indications.

Having decided that the case before us is one of genuine asthma, in the clinical sense, the first question is whether we can discover any exciting cause for the paroxysms. This may be found in bronchial irritation, which is by far the commonest cause, or in digestive derangement, which is relatively rare, or in reflex irritation

* Nothnagel's Encyclopædia of Practical Medicine, Art. Asthma.

from other organs—*e.g.* the genito-urinary—which is quite exceptional. Some asthmatic patients are much benefited by care as regards exposure, on the one hand, or by simple diet and the avoidance of late meals on the other. Often no exciting cause for the paroxysms can be discovered.

Of the many drugs which have been employed in the treatment of asthma, I should without hesitation give the first place to iodide of potash. I do not understand the view of those writers who simply mention this drug amongst a host of other remedies which may sometimes be found useful. In my opinion, it stands alone, and is often of signal value.* I prescribe it with some confidence, and have seen many cures from its use. Iodide of potash must, however, be given with a free hand. Five grains thrice daily is not too much, and even young children will tolerate this amount without inconvenience. The dose may require to be increased to ten, fifteen, or twenty grains thrice daily. I have not often had trouble with "iodism," and my experience is that if the patient has any susceptibility to the action of the drug the amount of the dose has little weight. A patient who will bear one-grain doses will usually bear much larger quantities. Small doses seem to have no influence over asthmatic paroxysms. The remedy should, I think, be given when the digestive organs are as empty as possible.

Next to the iodide of potash I should place arsenic, and the two remedies may often be usefully combined. The usual doses of Fowler's solution (3-8 m) are sufficient.

I have used lobelia somewhat largely in the treatment of asthma, and I am inclined to think that a small dose

* Dr. Little of Dublin, I know, shares this view.

of the ethereal tincture added to the iodide mixture sometimes heightens its efficacy.

I cannot from my own experience endorse the high place which some observers assign to strychnine and nux vomica in the treatment of asthma. These remedies are said sometimes to prove very efficacious in the neurotic type of asthma.

I have tried *grindelia robusta* occasionally, but have found it a very uncertain remedy.

I have seen one remarkable case where caffeine exhibited specific virtues, when other remedies had failed. So far, this case is an isolated one in my experience.

Other remedies, of which I have little personal experience and nothing definite to report, are chloral, bromide of potassium, *ipecacuanha*, *cannabis indica*, conium, pilocarpine, nitrite of amyl.

For the immediate relief of the paroxysms, the various powders and cigarettes containing stramonium and its congeners possess much potency, and many asthmatics find it difficult to dispense with their use. I cannot doubt, however, that they do in the main more harm than good, and that their continuous use is, in the long run, disastrous to the nervous and digestive systems. Many of my asthmatic patients have relinquished these drugs, finding the remedy worse than the disease. I very rarely prescribe these preparations, but I am often compelled to be accessory to their employment. Opium, cocaine, and chloroform should, I think, be avoided in the treatment of asthma. The danger of the formation of the opium or cocaine habit is great.

Inhalations have a definite, if very limited, value in the treatment of asthma. Hoffmann speaks highly of inhalations of warm water and steam.* With these may

* Nothnagel's *Encyclopædia of Practical Medicine*, Art. Asthma.

be combined such sedative remedies as compound tincture of camphor, compound tincture of benzoin, carbolic acid or creosote.

The potent, but erratic, influence of locality and climate upon asthma is well known. There are few subjects upon which it is more difficult to advise our patients. It is a matter of notoriety that locality has in some cases an overwhelming influence, either infallibly causing or inhibiting attacks. Some patients do best in the smoke-laden atmosphere of large towns, but this rule is not so general as has sometimes been asserted. Sea air, upon the whole, seems injurious, but there are many exceptions. In young patients with moderate bronchial symptoms and without any decided emphysema, the air of moderately elevated resorts seems sometimes useful. Mont Dore has acquired a special reputation in the treatment of asthma. Whether this is purely a matter of climate and elevation, or whether the arsenical waters of the locality have any influence, is a question. Some asthmatics do well at Davos [and St. Moritz, but upon the whole lower elevations are more desirable, *e.g.* Glion or Les Avants. If bronchial catarrh be a prominent feature and some emphysema be present, probably the preference should be given to warm and sheltered resorts, with sandy subsoil and, perhaps, the neighbourhood of pine woods. Sir Hermann Weber specially recommends Weissenburg, in Switzerland. Bournemouth or Woodhall Spa may have a trial.

It must be admitted that there are few more embarrassing problems than to be required to suggest a resort for an asthmatic. While not entirely a matter of guess-work, the indications are few and precarious. Perhaps this is of the essence of the case. When it is well known that patients may, in certain cases, either

precipitate or prevent attacks by a removal of a mile or two, or even from one side of the street to the other, it is evident that we are dealing with some factor which is quite incalculable in the present state of medical knowledge.

LECTURE XIV.

PHYSICAL EXAMINATION OF THE HEART.

SUMMARY.

Methods of examination, stethoscopes, the phonendoscope, posture of patient.

Inspection.

Shape of the præcordia, bulging, retraction, colour of superficial parts, areas of pulsation—normal and abnormal.

Palpation.

Character of the cardiac impulse, cardiac thrills, the *frémissement cataire*, systolic thrill in the aortic area, aneurismal thrill, systolic thrill in pulmonary area, diastolic thrill of patent ductus arteriosus, pericardial tactile fremitus.

Percussion.

Light and deep, mode of percussing, cardiac dulness, superficial and deep, causes of increase of cardiac dulness, causes of diminution of cardiac dulness.

Auscultation.

The cardiac sounds, variations in intensity, duration, tone rhythm.

Physiological variations, pathological variations, influence of activity of the heart's action, of posture, of the state of respiration.

Weakening of the first sound, intensification of the first sound, weakening and intensification of the aortic and the pulmonary second sound, Skoda's views, duration of the cardiac sounds.

Rhythm of the cardiac sounds, irregularity, intermittence, reduplication.

IN this and the following lecture we shall briefly consider the question of the physical examination of the heart.

Our treatment of the subject will necessarily be

somewhat summary. Only points of definite clinical importance will be dealt with.

When time permits, it is expedient to examine the patient both in the sitting (or standing) and in the recumbent position. Many murmurs are affected to an important extent by posture. If we are obliged to content ourselves with an examination in one position only, the recumbent position should always be preferred. No opinion should be given regarding the condition of the cardiac sounds until after auscultation with the patient recumbent. The patient's shoulders should be slightly raised.

Allowance should be made for the state of nervousness which is so often present, and in case of doubt a definite opinion should not be given until an examination of the heart can be made after the organ has quieted down.

Both the direct and the indirect methods of examination should be practised. The information obtained by the direct application of the ear to the chest (a thin handkerchief intervening) may be of the highest value. The form of stethoscope employed is of little consequence. On the whole, the binaural stethoscope—the simpler in form the better—is the more convenient. The phonendoscope is sometimes useful in exploring obscure murmurs.

We proceed by the following methods of examination viz. :—

- A. Inspection.
- B. Palpation.
- C. Percussion.
- D. Auscultation.

A. Inspection of the Heart.

We inspect—

- (a) The shape of the præcordia.

(b) The condition of the superficial parts.

(c) Areas of pulsation—normal and abnormal.

(a) *The shape of the præcordia.*

The præcordial region may be bulged or retracted.

Bulging of the præcordia is found in the following conditions :—

(a) Some cases of cardiac hypertrophy, most marked if it has developed in childhood when the chest-wall is soft and yielding. Pulsation is marked and heaving.

(b) Pericardial effusion. The intercostal spaces are full and widened, the impulse is feeble and fluttering, and is displaced upwards and outwards.*

(c) Aneurism of the heart or of the ascending aorta. The bulging in most cases is towards the base of the heart, and may be either to the right or to the left of the sternum, or in the episternal notch. Pulsation is usually present, but is not always perceptible to the eye.

Retraction of the spaces around the apex is usually considered an important sign of adherent pericardium.

(b) *The condition of the superficial parts.*

We may observe (1) Pallor, (2) Redness, (3) Cyanosis (4) Œdema, (5) An icteric tinge.

(1) *Pallor.*

The causes of pallor, other than those associated with cardiac disease, do not here concern us.

Pallor is characteristic of aortic regurgitation, and this point is one of considerable diagnostic importance.

* I need not enter into the question whether this impulse represents the true apex or not. This is doubtful.

Pallor usually accompanies conditions of syncope and collapse.

(2) *Redness.*

Abnormal redness of the skin is not a usual feature of cardiac disease. It may be found in connection with lesions of the sympathetic, or, as the result of the administration of amyl nitrite, atropine, oxygen. Abnormal redness of the lips is sometimes present in cardiac disease.

(3) *Cyanosis.*

The causes of cyanosis are many. We shall deal here only with the circulatory causes.

Cyanosis is especially characteristic of uncompensated mitral disease, regurgitation or stenosis, especially the latter. It may also occur in aortic disease, in myocarditis, in sclerotic or fatty degeneration of the heart muscle, in arterio-sclerosis, in "arterio-capillary fibrosis," and in pericarditis.

Cyanosis is often a prominent feature in congenital heart disease.

Sahli is of opinion that a certain degree of cyanosis accompanies even well-compensated mitral disease, whether regurgitation or obstruction.*

(4) *Œdema.*

Œdema is one of the most characteristic signs of cardiac disease. It usually begins, and is most marked, in the feet and legs. Sometimes it begins in the hands, and if the patient be bed-ridden it may make its first appearance in the loins. Exceptionally, it begins in the face.

Many patients have no œdema when in bed, but

* Lehrbuch der Klinischen Untersuchungs-Methoden, H. Sahli, Dritte Auflage, p. 25.

become œdematous in the feet and legs when moving about. Rest often has a marked effect on cardiac œdema.

(5) *Icteric tinge of conjunctivæ or skin.*

An icteric tinge may be observed in any form of heart disease accompanied by failing compensation.

It is most often seen in mitral disease, and indicates a considerable degree of passive congestion.

(c) *Areas of pulsation—normal and abnormal.*

In determining the position of the apex beat it is usually advisable to call in the aid of palpation, as well as inspection.

The apex beat is visible in most healthy persons in the fifth left interspace, about midway between the left parasternal line and the left mammary line.

In children the impulse may be somewhat higher, and more to the left than in the adult. In such cases it may be in the fourth interspace, and may be either in the left mammary line or even slightly external to it.

In old people, the beat may be in the sixth space.

The position of the apex beat presents certain physiological variations. By quiet respiration it is unaffected. By deep breathing it is raised, or in some cases made to disappear, owing to the extension of the expanded lung over the heart. When the patient turns on his left side the apex beat is moved somewhat to the left, when he turns on his right side to the right. The impulse is often more evident when the patient leans forward.

It is usually said that the area of visible pulsation under normal circumstances is about 2 cm. square, but there is no rule, and this statement cannot be relied upon.

In many healthy persons, especially those who are

deep-chested and have thick parietes, the apex beat is not visible.

The true apex is the lowest and most external part of the area of visible pulsation.

Displacements of the apex beat.

(a) Downwards.

e.g. Aneurism of the aorta, mediastinal tumour.
Emphysema, paralysis of the diaphragm,
Atonic relaxation of the great vessels.*

(b) Downwards and outwards.

e.g. Hypertrophy and dilatation of the left ventricle.
Hypertrophy and dilatation of the right ventricle.

(c) Outwards.

e.g. Hypertrophy and dilatation of the right ventricle.
Right pleural effusion.
Retraction of left lung from old pleurisy.
Adhesions of the heart in left pleurisy.
Pneumothorax on the right side.

(d) Inwards.

e.g. Left pleural effusion.
Retraction of right lung from old pleurisy.
Adhesions of the heart in right pleurisy.
Pneumothorax on the left side.

(e) Upwards.

e.g. Tumours, fluid or air in the abdomen.
Retraction of the lung from phthisis at left apex. The apex of the heart is tilted towards the left axilla.

* Walshe, Diseases of the Heart, fourth edition, p. 20.

- (f) Upwards and outwards.
e.g. Pericardial effusion.

Abnormal pulsation.

- (a) In the epigastrium. A certain slight degree of epigastric pulsation is common in health. A marked degree of pulsation in this region may be due to any of the following conditions:—

Hypertrophy and dilatation of the right ventricle.

Displacement of a normal heart downwards in consequence of emphysema or intra-thoracic tumour.

“Irritable” abdominal aorta.

Dilatation of the hepatic veins in tricuspid regurgitation.

Carcinoma of the pylorus.

Aortic regurgitation.

Abdominal aneurism.

- (b) At the left border of the sternum in the second interspace and below this point.

Pulsation in this region is probably due in nearly all cases to movements in the right ventricle, especially the conus arteriosus, and in the first portion of the pulmonary artery.

It may be due to an aneurism.

It is improbable that pulsation in this region is ever due to a hypertrophied left auricle. This is, however, a disputed point. I incline to the view that the left auricle, when hypertrophied and dilated, is displaced backwards, and does not cause any visible pulsation.*

* See, however, Sahli, *Lehrbuch der Klinischen Untersuchungs-Methoden*, p. 310.

- (c) Pulsation at the right border of the sternum in the second intercostal space and below this point.
Pulsation in this region may be due to—
Movements in a healthy aorta, uncovered by lung.
Dilatation of the aorta, especially in association with aortic regurgitation.
Dilatation and hypertrophy of the right ventricle,
Aortic aneurism.
Dilatation and hypertrophy of the right auricle (?).
- (d) Pulsation in the episternal notch.
Pulsation in this region may be due to—
Aneurism of the arch of the aorta.
Aortic regurgitation.
Functional palpitation.
- (e) Pulsation in the infraclavicular regions.
Pulsation in these regions may be due to—
Movements in the subclavian artery.
Aneurism of the aorta or some of its branches.
- (f) Pulsation in the distribution of the internal mammary artery.
Pulsation is sometimes visible in this area in cases of aortic regurgitation.
- (g) Pulsation in other areas.
Aneurism or mediastinal tumour may be suspected.

B. Palpation of the heart.

The hand should be first laid flat upon the left side of the thorax, and then the apical region should be explored more minutely. It is convenient to use the right hand for this purpose, if we stand before the patient, or the left hand, if we stand behind him.

By palpation we confirm or correct the information already obtained by inspection as to the position of the apex beat.

We obtain, further, additional information as to its character.

The impulse may be—

- (a) Weak, as normally in many thick-walled or deep-chested people, in persons of feeble vitality, in conditions of simple debility, in the exhaustion of fevers and other febrile diseases, in many forms of poisoning, in fatty heart, pneumothorax, emphysema, pleural effusion, mediastinal tumour, œdema of the thorax.
- (b) Weak and fluttering—as in pericardial effusion.
- (c) Quick, diffused, and wavy—as in cardiac dilatation.
- (d) Slow, heaving, and thrusting—as in cardiac hypertrophy.
- (e) Strong and slapping, but not heaving—as in functional palpitation, chlorosis, Graves's disease, abuse of tea and tobacco, certain stages of fevers.
- (f) Irregular in rhythm—as in functional palpitation, some forms of organic heart disease, some brain diseases, states of exhaustion, tachycardia, and many other conditions.

By palpation we detect the presence of various cardiac thrills.

Cardiac thrills.

The most important of these is the well-known pre-systolic thrill (*frémissement cataire*) of mitral stenosis, first noticed by Corvisart, afterwards more fully studied by Lænnec.

The latter's description of this interesting sign was as follows :—

“ Le frémissement cataire est une sensation particulière que perçoit la main appliquée sur la région du cœur, et que l'on a comparée au frémissement qui accompagne le

murmure de satisfaction que font entendre les chats quand on les caresse. Ce phénomène accompagne constamment le bruit de râpe, et indique comme lui un obstacle mécanique apporté au cours du sang par le rétrécissement de quelqu'un des orifices du cœur."

Lænnec did not recognize the close association of this sign with mitral stenosis, and he was of opinion that it was frequently found apart from organic lesion of the heart *—an opinion which does not seem to be well founded.

The "presystolic thrill," or "*frémissement cataire*," is usually felt over a considerable area in the apical region. It may sometimes be felt throughout the whole of diastole—oftener it is confined to the latter part of diastole, *i.e.* it is truly presystolic in rhythm. In the former case it is not of equal intensity throughout the whole of diastole, but is weak at the beginning of diastole and increases to a maximum towards its close. It is abruptly terminated by the cardiac impulse. The classical comparison of this thrill to the sensation conveyed to the hand when placed upon the back of a purring cat is a singularly apt and expressive one. When well marked this thrill may be regarded as pathognomic of mitral stenosis, and in its typical form it probably indicates a considerable degree of obstruction and decided hypertrophy of the left auricle. I adopt the theory of its causation usually associated with the name of Gairdner.

This thrill, like the corresponding murmur, often varies much from day to day, and may disappear during rest to reappear after exertion. The relation of the thrill to the murmur is close.

Systolic thrill in the aortic area.

This thrill is common in aortic obstruction. It is rather of the nature of a vibration, and does not possess

* *L'Auscultation Médiate*, tome iii. p. 67.

the "purring" quality of the pre-systolic thrill. It is sometimes very intense.

It is felt chiefly at the upper part of the sternum, in the second right intercostal space, or at the episternal notch, but may be recognizable over the whole chest.

Systolic thrill in the pulmonary area.

This thrill is sometimes present in congenital obstruction of the pulmonary artery.

Aneurismal thrill.

This is a common form of thrill. It is systolic in time. Its seat depends upon the locality of the aneurism.

Thrill depending on patency of the ductus arteriosus.

This thrill is usually felt in the second left interspace. It is diastolic in time.

A diastolic thrill in the aortic area depending upon aortic regurgitation has not been common in my experience.

Pericardial tactile fremitus.

This is a rubbing sensation often felt over the præcordial area in pericarditis.

It may be absent even when the pericardial friction sound is well marked.

It may co-exist with a considerable effusion.

Diastolic shock.

The diastolic shock which often accompanies aortic aneurism is well known.

Fluctuation.

In certain cases of pericardial effusion fluctuation can be made out.

C. Percussion.

Percussion throws light upon the size and relations

of the heart. In interpreting percussion signs it is indispensable to have constant regard to the state, not only of the heart, but of the adjacent organs, especially the lungs.

Light percussion is to be preferred. One finger may usually with advantage be used as a hammer, and the "staccato" stroke will generally be found best. Light percussion is requisite whether we seek to map out the "superficial" or the "deep" cardiac dulness. I may assume that you are familiar with the boundaries of these areas. The distinction between "superficial" or "absolute" cardiac dulness and "deep" or "relative" cardiac dulness is neither easy to define with accuracy nor always logically valid. Much depends upon the strength of the percussion stroke. Guttman denies that the right edge of the heart can be defined by percussion.* Gibson affirms that it can.† In some cases, at least, the entire sternum gives a resonant note. Gee states that in passing from the heart to the liver a heightening of pitch in the percussion note, and an increase of resistance, may sometimes be made out, and that sometimes there is a distinct band of faint resonance between the two organs.‡

The measurements of superficial cardiac dulness in a person of average size are $3\frac{1}{2}$ inches transversely, from the mid-sternal line; and 2 to $2\frac{1}{2}$ inches vertically along the left border of the sternum (T. D. Savill).

The area of cardiac dulness is increased in the following conditions :—

Hypertrophy.

Dilatation.

Pericardial effusion.

* Handbook of Physical Diagnosis, Napier's translation, p. 254.

† G. A. Gibson : Diseases of the Heart and the Aorta, p. 137.

‡ S. Gee : Auscultation and Percussion, third edition, p. 91.

Aneurism of the heart or the aorta.

Dilatation of the aorta.

Mediastinal tumour.

Retraction of the lung from old pleurisy or from phthisis.

Practically, hypertrophy and dilatation are always associated. In proportion as hypertrophy of the left ventricle is well marked, the increased area of dulness will be downwards and outwards. If dilatation of the left ventricle be the main feature, the increase of dulness will be chiefly outwards.

Dilatation and hypertrophy of the right ventricle sometimes cause an increase of dulness to the right only, sometimes also to the left.

Dilatation and hypertrophy of the right auricle cause an increase in dulness upwards and to the right.

Pericardial effusion produces a cone or pear-shaped area of dulness, with the base below and the apex above. Dulness may extend as high as the top of the sternum, as low as the tip of the ensiform cartilage. There is dulness in the fifth right intercostal space (Rotch). The right border of the dulness slopes upwards and towards the left, while in hypertrophy and dilatation the right border of dulness is a vertical line.

The other cases of increased area of dulness do not call for any special comment.

The area of cardiac dulness is diminished in the following conditions—

Emphysema.

Pneumothorax of the left side.

Pneumo-pericardium.

Atrophy of the heart (?).

In emphysema the cardiac dulness may be entirely annulled.

D. Auscultation.

In practising auscultation of the heart we should take the cardiac areas in succession—aortic, mitral, pulmonary, and tricuspid—and in each area put the questions—

(a) What of the first sound ?

(b) What of the second sound ?

(c) What of the long pause ?

The short pause may be neglected.

The cardiac sounds.

It is of the utmost importance to familiarize the ear thoroughly with the normal cardiac sounds. These present considerable variations, and the most difficult problem in cardiac auscultation is often to decide—Is the sound present in this case within the normal physiological limits or not? This question can be answered with confidence only when the possible physiological variations are thoroughly familiar to the auscultator.

You are, no doubt, familiar both with the actual position of the cardiac valves and with the points in the surface of the chest where the sounds produced at the various orifices are heard with maximum intensity.

It is not always easy to decide which is the first sound and which the second sound. In case of difficulty we may obtain assistance from the following considerations:—

(a) Attention to the accent.

In the mitral and the tricuspid areas the accented sound is the first sound.

At the base of the heart and over the great vessels the accented sound is the second sound.

(b) Attention to the pause.

The short pause follows the first sound ; the long pause follows the second sound.

If the action of the heart be extremely rapid this distinction may be difficult to observe.

(c) Comparison of the sounds with the cardiac impulse.

The moment of the impulse marks the commencement of the ventricular systole, and is coincident with the first sound.

The carotid and the radial pulse are not to be relied upon in this connection.

(d) Attention to certain murmurs, e.g. a typical pre-systolic murmur determines the moment of systole, and hence of the first sound.

The sounds of the heart may present variations as regards—

(a) Intensity.

(b) Timbre.

(c) Pitch.

(d) Duration.

(e) Presence or absence of murmur.

Variations in the intensity of the cardiac sounds.

These variations depend partly upon the condition of the heart itself, partly upon the condition of parts surrounding the heart. They may be either physiological or pathological.

Physiological variations in the intensity of the cardiac sounds.

These depend upon the following considerations :—

(a) Position.

The first sound is louder at the apex, the second sound at the base. The second sound may, under normal conditions, be louder either in the aortic or the pulmonary

area.* On the whole, it is more often louder in the aortic area in the elderly, louder in the pulmonary area in the young and middle-aged. It is a common error to attach great importance to slight differences in the intensity of the second sound in these areas. In a large proportion of cases these differences have no pathological significance.

(b) *The condition of the parietes.*

Thickness of the thoracic wall, whether due to muscular development, fat, or occasionally to œdema, produces a notable weakening in the intensity of the heart-sounds. It is often remarkable how weak the sounds are in muscular, well-developed men. Exceptional mammary development in women has a similar effect.

In thin people the sounds, *cæteris paribus*, are intensified.

(c) *Posture.*

The heart-sounds are louder in the erect than in the recumbent position.

(d) *Activity of the heart's action.*

The sounds usually become louder and more distinct as the heart's action becomes more vigorous—as during exercise or excitement.

(e) *Respiration.*

The sounds are more intense during expiration and during the respiratory pause than during inspiration, inasmuch as during inspiration the margins of the lung creep forward and cover the heart.

Pathological variations in the intensity of the heart-sounds.

The heart-sounds are intensified in the following conditions:—

* On this subject see Guttman, *Handbook of Physical Diagnosis*, p. 272 ; Broadbent, *Heart Disease*, p. 181 ; Gibson and Russell, *Physical Diagnosis*, p. 45.

- (a) Consolidation of the lungs.
- (b) Excavation of the lungs.
- (c) Pneumo-pericardium.
- (d) Gaseous distension of the stomach.
- (e) Some cases of dilatation of the heart.

The heart-sounds are weakened in the following conditions :—

- (a) Debility, wasting diseases, later stages of pyrexial conditions.
- (b) Degeneration of heart-muscle, *e.g.* fatty heart.
- (c) Syncope and collapse.
- (d) In connection with certain valvular defects.
- (e) Pericardial effusion.
- (f) Emphysema.

We shall now consider the question of variations in intensity with regard to the different sounds.

The first sound.

The first sound as heard at the apex is intensified in the following conditions :—

- (a) Certain febrile states.
- (b) Neurotic disturbance.
- (c) Mitral obstruction.
- (d) Some cases of dilatation of the left ventricle.
- (e) Some cases of interstitial nephritis.

The first sound as heard at the apex is weakened in the following conditions :—

- (a) In the later stages of pyrexial conditions, *e.g.* typhoid fever. The total disappearance of the first sound in such conditions is of grave augury.
- (b) In wasting diseases, debility, and cachectic conditions generally. There are many exceptions to this rule. It must be borne in mind that wasting diseases produce thinning of the thoracic parietes,

and this tends to intensification of the cardiac sounds. We are sometimes surprised by the intensity of the cardiac sounds in thin and debilitated people.

- (c) In myocarditis and fatty heart.
- (d) In some cases of mitral regurgitation.
- (e) In some cases of hypertrophy of the left ventricle, *e.g.* in granular kidney.

The relation of the intensity of the first sound of the heart to cardiac hypertrophy and cardiac dilatation is a very important practical point. In general, the first sound is muffled and weakened (but prolonged) in hypertrophy; while it is intensified (but shortened) in dilatation. This statement, which rests upon clinical observation, is most easily explained on the supposition that the first sound is mainly due to the tension of the auriculo-ventricular valves. The sound produced by the tension of the valves is well conveyed through the thin ventricular wall of a dilated heart, ill-conveyed through the thick ventricular wall of a hypertrophied heart.

The second sound.

The aortic second sound is intensified in the following conditions :—

- (a) Dilatation of the aorta.
- (b) Aneurism of the aorta.
- (c) Arteric-sclerosis.
- (d) Renal disease.
- (e) Many cases of hypertrophy of the left ventricle.

Several of the above causes may co-exist.

Intensification of the aortic second sound indicates in most cases increased tension in the aorta.

The aortic second sound is weakened in the following conditions :—

- (a) Aortic obstruction.
- (b) Mitral disease.
- (c) Weak or degenerated left ventricle.
- (d) Relaxation of the arterioles.

The pulmonary second sound is intensified in the following conditions :—

- (a) Emphysema.
- (b) Fibrosis of the lung.
- (c) Mitral disease.

The pulmonary second sound is weakened in the following conditions :—

- (a) Weak or degenerated right ventricle.
- (b) Tricuspid regurgitation.

In aortic regurgitation the second sound, as heard in the aortic area, at midsternum, along the edges of the sternum, or at the ensiform cartilage, is more or less weakened and obscured by murmur; but as heard over the great vessels of the neck is often loud and ringing, a point of prognostic importance.*

To Skoda we owe the importance which since his day has been attached to the intensification or "accentuation" of the pulmonary second sound as a sign of compensated mitral disease. His statement of this point is as follows :—

"When the pulmonary artery is abnormally distended, which it always must be when the pulmonary circulation is obstructed, its second sound becomes very loud, whilst the aortic second sound is either weak or inaudible or is replaced by a murmur. The pulmonary artery, when thus distended, presses with increased force upon the blood within it, and consequently the backward stroke of the columns of blood against its semilunar valves becomes more forcible." †

* W. H. Broadbent : *Heart Disease*, third edition, p. 161.

† J. Skoda : *Auscultation and Percussion*, Markham's translation, p. 202.

In spite of the great weight of authority which may be quoted in support of this doctrine, I cannot help questioning its accuracy upon clinical grounds. It may be remarked—

- (a) That we have little certain knowledge of the conditions of tension in the pulmonary circuit, and that the fundamental postulates on which Skoda's doctrine rests are by no means indisputable.
- (b) That the "accentuation" of the pulmonary second sound, although undoubtedly common in cases of mitral disease, is sometimes present in thin people in whom there is no reason to suspect increased tension in the pulmonary circulation.
- (c) That "accentuation" of the pulmonary second sound is not constant in compensated mitral disease.*

It is generally held that the "accentuation" of the pulmonary second sound is indicative of well-compensated mitral disease, and that it disappears when tricuspid regurgitation is set up.

It is also usually taught that this "accentuation" is present in emphysema.

The timbre of the cardiac sounds.

This is a difficult subject, upon which it is impossible to lay down definite rules, capable of clinical application. The timbre of the cardiac sounds depends upon structural peculiarities in the valves or the heart-muscle, which may not involve any actual lesion. It also depends in part upon the condition of surrounding organs.

The ringing quality of the aortic second sound in many

* On this difficult subject see the following: Andrew, St. Bartholomew's Hospital Reports, vol. i. p. 13; Gee, Auscultation and Percussion, p. 207; O'Carroll, "On Accentuation of the Second Sound in the Pulmonary Area," *Dublin Journal of Medical Science*, September, 1900.

cases of arterio-sclerosis is a case where peculiarities in timbre may possess diagnostic importance.

Obscured or muffled timbre often precedes the development of a murmur.

The pitch of the cardiac sounds.

Variations in pitch of the cardiac sounds can often be appreciated, especially by the musically trained ear, but we cannot at present make any definite clinical applications of this point.

The loud first sound in mitral stenosis, and the so-called "accentuated" pulmonic second sound often possess a heightened pitch.

In neurotic excitement the pitch of the first sound may be raised.

The duration of the cardiac sounds.

The prolonged first sound of hypertrophy and the short first sound of dilatation are characteristic.

Alterations in the duration of the cardiac sounds are apt to be obscured by murmur. Thus in aortic stenosis there is a prolonged first sound, but it is usually lost in the accompanying murmur.

The rhythm of the cardiac sounds.

As the question of the rhythm of the cardiac sounds is a subject of much complexity and importance, we shall devote a lecture specially to it. (See Lecture XVIII., Disorders of the Cardiac Rhythm.)

LECTURE XV.

PHYSICAL DIAGNOSIS OF CARDIAC DISEASE (CARDIAC MURMURS).

SUMMARY.

Murmurs are—

A. Pericardial.

B. Endocardial.

A. Pericardial Friction.

Pleuro-pericardial friction.

Pericardial splashing.

B. Endocardial Murmurs.

Their mechanism.

(*a*) Quality of murmurs.

(*b*) Point of maximum intensity.

(*c*) Line of conduction.

(*d*) Time.

Mode of timing murmurs.

Systolic, diastolic, and pre-systolic murmurs.

Varieties of systolic murmur, and their differential diagnosis.

Diastolic murmurs, varieties, and differential diagnosis.

Pre-systolic murmurs, varieties, and differential diagnosis.

(*e*) Influence of posture and movement.

Cardiac Murmurs.

Cardiac murmurs are either (*a*) Pericardial, or (*b*) Endocardial.

The term “murmur” is not a convenient one to describe the friction sound of pericarditis, but it has the sanction of authority.

Pericardial Friction.

Pericardial friction has the following characters: It is in most cases rough, rubbing, creaking, grating or rasping

in quality. Exceptionally it is soft, and simulates an endocardial murmur. It is "superficial." It has usually a "to and fro" quality.

The intensity of the sound varies, but is usually greater than that of pleural friction. Its loudness has a relation to the energy of the heart's action. It is sometimes loudest when the breath is held at the end of a deep inspiration. It is often louder in the sitting than in the recumbent position. Its loudness is sometimes increased by the pressure of the stethoscope. It may be heard over any part of the præcordia. It is commoner at the base than at the apex. Walshe thought it was most frequent in the third interspace.* It may accompany any phase of the cardiac cycle, but is usually heard with the ventricular systole and diastole. It has no fixed or definite relation to the cardiac sounds. This is the most important point of all with regard to the differential diagnosis. It is not definitely propagated in any direction, and is usually at once lost in shifting the stethoscope from the præcordial area. It is not heard over the great vessels of the neck. In some cases, however, of very loud pericardial friction the sound is heard over a wide area.

Pericardial friction may continue in the presence of a considerable effusion.

Pleuro-pericardial Friction.

This sound is produced in certain cases by rubbing of the costal or pulmonary pleura against the pericardial pleura. It is most audible during inspiration. It is usually annulled by holding the breath. It is heard outside the usual limits of audibility of pericardial friction.

Pericardial Splashing.

In certain rare cases where fluid and gas are both

* Diseases of the Lungs, fourth edition, p. 108.

present in the pericardial sac a sound is heard comparable to the succussion-splash of pneumothorax.

Endocardial Murmurs.

Endocardial murmurs are due to vibrations or tremblings in the heart or vessels depending on eddies in the blood-stream. Much has been written upon the mechanism of heart murmurs, but theoretical questions of this kind lie outside the scope of these lectures. I can only glance at the subject. The most usual cause of vibrations in the heart or vessels is the formation of "fluid veins" in the circulating blood, due to some impediment to the even onward flow of the blood.

It is not likely that alterations in the quality of the blood ever give rise to murmurs.

It is generally thought that a murmur may be caused by roughening of a valve or of the endothelium of the arteries; but Chauveau's experiments throw doubt on this doctrine. The mechanism of "hæmic," "functional," or "accidental" murmurs is not known. There are many theories on the subject, but no certain knowledge.

Murmurs are occasionally present in conditions of perfect health.

Murmurs are usually audible only to the listening ear. In exceptional cases they are audible to the patient also.

In investigating an endocardial murmur we attend to the following points:—

- (a) The quality of the murmur.
- (b) Its point of maximum intensity.
- (c) Its line of conduction.
- (d) Its time.
- (e) The effect of posture and movement.

A. The Quality of Murmurs.

The quality of murmurs is variable, apt to change from day to day, more or less under the influence of rest, exercise, or excitement, may easily mislead. Nevertheless, I cannot agree with those writers who regard the quality of murmurs as of no practical importance. Gee's statement, "Murmurs were once characterized according to their acoustic qualities, whether blowing, filing, rasping, sawing ; but these are vain distinctions : in order to render murmurs serviceable in the diagnosis of disease we now regard only two of their properties, namely, their place and time," * seems to me to overstate the case. It is easy to over-estimate the importance of the quality of cardiac murmurs, but we cannot wholly ignore this point without loss. The rough, vibratory, "ingravescent" quality of the murmur of mitral obstruction, the blowing "bellows" quality of the murmur of mitral regurgitation, the prolonged and generally low-pitched quality of the murmur of aortic regurgitation are too common to be ignored, frequent though the exceptions to these rules may be. The quality of a murmur may give us an important hint towards a diagnosis. It does not do more. The precise determination of the origin and significance of a murmur always depends upon other considerations.

Recognizing, then, fully the limited value of the quality of cardiac murmurs, we may study this point with some advantage.

The "blowing" murmur (*bruit de soufflet*) is the commonest form of murmur. It is characteristic of mitral regurgitant murmurs and of "hæmic," "functional," or "accidental" murmurs ; it is common in aortic regurgitation, less common in aortic stenosis, rare in mitral stenosis. It is common over the sac of an aneurism.

* Auscultation and Percussion, third edition, p. 155.

The "rough vibratory" murmur is most often heard in aortic stenosis and in mitral stenosis.

The "crescendo," or "ingravescent" murmur, *i.e.* the murmur which begins softly, but rapidly increases to a maximum, is very characteristic of mitral stenosis. It may also be heard in tricuspid stenosis. It is to be noted that in contra-distinction to this murmur, most cardiac murmurs are "diminuendo" in quality.

The prolonged ringing murmur is sometimes heard in aortic regurgitation.

The "cooing" or "whistling" murmur is sometimes present both in aortic and in mitral disease. It may be due to some peculiar narrowing of the orifice, or to the floating of a filament of fibrin in the blood-stream.

Musical and scraping murmurs are nearly always organic.

The intensity or loudness of a murmur is closely related to the energy of the heart's action. It is no index of the extent of the lesion. Cases with a very loud murmur often do well, while a weak, fugitive murmur, varying with successive beats, is often of very evil augury. In the worst cases of all the murmur may entirely disappear. The re-appearance of a definite murmur after it has been for some time inaudible is often coincident with improvement in the patient.

The intensity or loudness of a murmur is of no importance as a help in discriminating functional from organic murmurs. A functional murmur is often weak, but may be loud. An organic murmur is either loud or weak.

The pitch of murmurs is variable.

The murmur of aortic regurgitation is usually, in my experience, of low pitch.

Obstructive murmurs are usually of higher pitch than regurgitant.

B. The Point of Maximum Intensity of Murmurs.

Aortic stenosis.

The point of maximum intensity of the systolic murmur of aortic stenosis is usually at the aortic cartilage or in the second right interspace or over the manubrium sterni. It may also be loudly heard over the carotids and over the left ventricle, including the apex, but perhaps never has its maximum at these points.

Aortic regurgitation.

The point of maximum intensity of the diastolic murmur of aortic regurgitation is very variable.

It is not usually found at the aortic cartilage, or in the second right interspace.

Oftener it is at midsternum about the level of the third costal cartilage, or along the left or the right border of the sternum, as low as the fourth interspace, occasionally in the neighbourhood of the ensiform appendix, perhaps never at the apex.*

This murmur is usually audible, though hardly ever attains its maximum, in the aortic area, but it may be entirely absent there, although loudly heard along one or other edge of the sternum.

Mitral obstruction.

The point of maximum intensity of the pre-systolic murmur of mitral obstruction is at, or a little inside, the apex.

The pure diastolic murmur, sometimes present in this condition, has its maximum a little higher up, over the left auricle.†

Mitral regurgitation.

The point of maximum intensity of the systolic

* Broadbent denies that this murmur is ever loudest at the apex: *Heart Disease*, third edition, p. 149.

† Sahli, *Lehrbuch der Klinischen Untersuchungs-Methoden*, p. 269.

murmur of mitral regurgitation is in the great majority of cases at the apex. According to many authorities—Naunyn, Balfour, Leube, Sahli, and others—this murmur has sometimes its maximum at the upper left edge of the sternum, in the neighbourhood of the left auricle. Broadbent says, "Occasionally the murmur of mitral regurgitation is heard more loudly in the fourth or even in the third space in the vertical nipple line, or just outside it, than at the apex."* On the other hand, Graham Steel calls this doctrine "a great heresy."†

This is a very doubtful point.

It is not certain that the murmur present in these cases in the pulmonary area is really the same murmur which is audible at the apex.

Tricuspid obstruction.

The point of maximum intensity of the pre-systolic murmur of tricuspid obstruction is at the lower end of the sternum, or a little to the left or right of this point.

Tricuspid regurgitation.

The point of maximum intensity of the systolic murmur of tricuspid regurgitation is at the lower end of the sternum, or a little to the left or right of this point.

Pulmonary obstruction.

The point of maximum intensity of the systolic murmur of pulmonary obstruction is in the second left interspace near the sternum or at the third left intercostal cartilage.

Pulmonary regurgitation.

The point of maximum intensity of the diastolic murmur of pulmonary regurgitation is in the second left

* Heart Disease, p. 173.

† Encyclopædia Medica, vol. iv. p. 421.

interspace near the sternum, or below that point along the left edge of the sternum, sometimes as low as the ensiform cartilage.

Hæmic, functional, or accidental murmurs.

These murmurs, which are always systolic in time, have their maximum intensity usually in the second left interspace, sometimes over the right ventricle, rarely in the aortic or mitral areas.

C. Line of Conduction of Murmurs.

The conduction of murmurs depends upon the conducting power of the heart substance, the direction of the blood-stream at the moment when the murmur is generated, the state of the thoracic parietes, and the condition of neighbouring organs, especially the lungs. Conduction is promoted by thinness of the parietes and by pulmonary consolidation. It is impeded by thickness of parietes and by emphysema.

Aortic stenosis.

The murmur of aortic obstruction is usually well conducted into the large vessels of the neck—a point which in case of doubt will go far to distinguish it from the murmur of mitral regurgitation.

This murmur is often conducted over the left ventricle, and may be well heard at the apex.

Aortic regurgitation.

The murmur of aortic regurgitation is conducted downwards along one or both edges of the sternum, often to the ensiform cartilage, exceptionally to the apex.

It is also conducted upwards into the large vessels of the neck, but this, in my experience, is the exception rather than the rule.

Mitral obstruction.

As a rule, the conduction of the pre-systolic murmur of mitral obstruction is very limited.

It is usually conducted for a limited space towards the left border of the sternum, or slightly upwards, exceptionally over a considerable area of the præcordia, very rarely outside the apex, towards the axilla or to the back.

Mitral regurgitation.

As a rule, the conduction of the murmur of mitral regurgitation is somewhat wide.

It is usually conducted into the left axilla, often to the back, occasionally towards the base, exceptionally all over the præcordial area.

It is never conducted into the large vessels of the neck.

The conduction of this murmur to the back is by direct conveyance of vibrations from the left ventricle to the dorsal vertebræ.

Tricuspid stenosis.

This very rare murmur is said to be conducted upwards and towards the left.

Tricuspid regurgitation.

This murmur is conducted upwards, either to the left or to the right.

Pulmonary obstruction.

The systolic murmur of pulmonary obstruction is conducted along the branches of the pulmonary artery, and may sometimes be audible over the whole præcordia, or between the shoulder-blades. It is not usually audible in the neck.

It is often well conducted under the left clavicle, a point which serves to differentiate it from the murmur of aortic obstruction.

Pulmonary regurgitation.

This very rare murmur is conducted downwards along the sternum. It is not audible in the neck.

Hæmic, functional, or accidental murmurs.

These murmurs are not well conducted in any direction, as a general rule, but exceptions occur.

D. The Time (rhythm) of Murmurs.

Murmurs as regards their time are :—

(a) Systolic.

(b) Diastolic.

(c) Pre-systolic.

Systolic murmurs are synchronous with the systole of the ventricles. They accompany or replace the first sound.

The term post-systolic is used by some writers to indicate a murmur which immediately succeeds the systole of the ventricles, "tails off," as it were, from the first sound, and is continued into the systolic pause.

Diastolic murmurs are synchronous with the diastole of the ventricles. They accompany, replace, or follow the second sound.

The term pre-diastolic is used by some writers in the same sense as post-systolic.*

Pre-systolic murmurs are synchronous with the systole of the auricles, and occur just before the systole of the ventricles.

Pre-systolic murmurs are sometimes called auricular systolic, sometimes diastolic murmurs. They run up, as it were, into the first sound, and are at once terminated by it.

Systolic murmurs.

Systolic murmurs are the commonest of all murmurs. They may be heard in the following conditions :—

* *E.g.* Sahli, *op. cit.* p. 273.

Aortic obstruction.

Roughening of the aortic valve segments.

Dilatation of the aorta.

Roughening of the intima of the aorta.

Acute or subacute aortitis (Broadbent).

Mitral regurgitation.

Tricuspid regurgitation.

Pulmonary obstruction.

Patent foramen ovale.

Anæmia and debility.

Aneurism.

Pressure on a vessel by a tumour or enlarged gland.

Compression of the edge of the lung by the ventricular systole.

The differential diagnosis of some of the above murmurs may be easy ; in other cases, great—even insuperable—difficulties may be encountered.

We shall proceed to consider the most important cases.

Systolic murmur in the aortic area.

It is of the first importance to recognize that this murmur is only in extremely rare cases due to definite obstruction of the aortic orifice.

A systolic murmur in the aortic area is one of the commonest of murmurs ; aortic stenosis is one of the rarest of lesions.

The causes of a systolic murmur in the aortic area are as follows :—

Aortic obstruction.

Roughening of the segments of the aortic valves.

Dilatation of the aorta.

Roughening of the intima of the aorta.

Acute or subacute aortitis.

Anæmia and debility.

Some of the above conditions cannot be definitely recognized by our present clinical methods.

The most important point to decide is whether aortic obstruction is present or not. The diagnosis of this lesion is not difficult. In aortic stenosis we may expect to find the following signs :—

- (a) The murmur is usually loud, prolonged, and rough ; its point of maximum intensity is in the second right intercostal space or over the adjacent portion of the sternum ; it is well conducted into the large vessels of the neck, sometimes, also, over the left ventricle and to the apex ; it is sometimes audible over the whole præcordial area and over the back ; the second sound at the aortic cartilage is weak or inaudible.
- (b) The left ventricle is usually moderately hypertrophied, never to the extreme degree common in aortic regurgitation. A systolic thrill is sometimes present.
- (c) The pulse is usually small, of good tension, regular, slow, and somewhat infrequent.

Exceptions to the above rules may be met with. The murmur may be soft, blowing, or short. The left ventricle may exhibit no hypertrophy,* the pulse may depart from its typical characters. Where the left ventricle undergoes decided dilatation, and where secondary mitral regurgitation is set up, it is evident that the physical signs will undergo important alterations.

The two conditions most apt to be confused with aortic stenosis are :—

- (a) Roughening of the intima of the aorta.
- (i) Anæmia and debility.

The former condition can usually be excluded by attention to the characters of the pulse.

* Traube.

The latter condition can usually be excluded by attention to the point of maximum intensity of the murmur (the murmur of anæmia and debility, though often heard at the aortic cartilage, has very rarely its maximum in this situation), to the characters of the pulse, to the general constitutional symptoms, and to the history.

The systolic murmur of mitral regurgitation.

This murmur has the following characters: It is in most cases blowing in quality, soft or harsh, loud or weak; its point of maximum intensity is at the apex, or a little above that point; its line of conduction is usually to the left axilla, exceptionally to the base or all over the præcordia; it is often well heard posteriorly between the vertebral border of the left scapula and the spine; it is not audible over the large vessels of the neck; the first sound may be well-marked, weak, or absent; the pulmonary second sound is often, not always, "accentuated;" the murmur is often affected by posture, being usually louder when the patient is recumbent.

In certain cases this murmur is "post-systolic"—*i.e.* we hear a clear and well-marked first sound which at once shades off into a blowing murmur. It has been supposed that this type of murmur indicates that the valve first shuts but immediately gives way before the completion of the ventricular systole.*

Accompanying this murmur we find in most cases evidence of hypertrophy and dilatation of the left ventricle, and the left auricle, often similar changes on the right side of the heart, a somewhat small, low-tensioned, occasionally irregular, and usually somewhat frequent pulse.

The changes in the chambers of the heart and the

* Broadbent: Heart Disease, third edition, pp. 44-45.

characters of the pulse depend upon the degree of compensation present.

The murmurs most apt to be mistaken for the murmur of mitral regurgitation are the following :—

- (a) The murmur of aortic stenosis.
- (b) The murmur of tricuspid regurgitation.
- (c) Certain cardio-pulmonary murmurs.

I reserve for consideration afterwards the murmur of anæmia and debility, sometimes heard in the mitral area, which is really a particular case of mitral regurgitation.

The murmur of mitral regurgitation may be distinguished from that of aortic stenosis by attention to its point of maximum intensity, its line of conduction, and the characters of the pulse. The quality of the murmur may sometimes assist the diagnosis. In mitral regurgitation the murmur has, in the great majority of cases, its point of maximum intensity at the apex. This is rarely, if ever, the seat of maximum intensity of the murmur of aortic obstruction. The murmur of mitral regurgitation is never conducted into the large vessels of the neck; the murmur of aortic stenosis is almost invariably conducted thither. The pulse in mitral regurgitation is low in tension, frequent, and sometimes irregular. The pulse of aortic stenosis is of good tension, infrequent, and regular.

The murmur of mitral regurgitation may be distinguished from that of tricuspid regurgitation by attention to its point of maximum intensity and the state of the venous circulation. In tricuspid regurgitation the murmur has its maximum at the lower end of the sternum, and there is visible pulsation in the veins.

The murmur of mitral regurgitation may require to be distinguished from that depending upon cardio-pulmonary causes. This latter murmur is much influenced by respiration, is usually intensified by inspiration, and weak or

absent during the respiratory pause. We shall return to this point.

The systolic murmur of tricuspid regurgitation.

This murmur is soft in quality, its point of maximum intensity is at the lower end of the sternum, or a little to the left or to the right of that point ; it is sometimes heard all over the right ventricle ; it may be heard over the right auricle ; it is usually audible at the apex. "When a murmur heard at the apex is lost immediately to the left of the beat, while it is audible between the apex and the lower end of the sternum, its seat of production is at the tricuspid and not at the mitral orifice." (Broadbent.)

It was formerly thought that this was a rare murmur ; it is now believed to be common.

It is usually accompanied by visible pulsation in the jugular veins, internal and external, by evidence of mitral disease, pulmonary cirrhosis or emphysema, and by some degree of dropsy and cyanosis.

Epigastric pulsation from congestion of the hepatic veins or from dilatation and hypertrophy of the right ventricle is usually present.

The systolic murmur of pulmonary obstruction.

This murmur is usually loud and rough, its maximum intensity is in the second or third left interspace near the sternum, it is conducted along the branches of the pulmonary artery, and is sometimes heard all over the chest, front and back. It is almost always congenital in origin, and is usually accompanied by other signs of congenital heart defect.

The systolic murmur of anæmia and debility (the so-called accidental murmur).

This is a most important murmur from the point of

view alike of diagnosis, prognosis, and treatment. Some of the most difficult questions in connection with cardiac diagnosis are concerned with the differentiation of this murmur. It is important to recognize that an "accidental" murmur may be present in the pulmonary area in perfectly healthy people.

This murmur varies much in quality. It is quite erroneous to suppose that it is always soft and weak. On the contrary, it may be loud and harsh, especially in the pulmonary area.

Its point of maximum intensity is in the great majority of cases in the pulmonary region, exceptionally over the right ventricle, and very rarely at the apex or in the aortic area.

The murmur is not well conducted.

Its time is almost invariably (perhaps always) systolic. It is much affected by exercise and posture. Sometimes it disappears during exercise; sometimes, on the contrary, it is heard only during exercise, or after exercise. It is generally louder in the recumbent than in the sitting or standing posture.

This murmur is often accompanied by signs of anæmia, by a venous hum in the neck, and by a pulse of somewhat good tension.

It is generally agreed that "accidental" or "hæmic" murmurs are almost invariably systolic in time. Leube denies that these murmurs are ever diastolic.* Strümpell affirms, however, that he has heard a loud diastolic murmur of hæmic origin in a case of pernicious anæmia.† Sahli is of opinion that in certain cases of extreme anæmia a diastolic murmur is sometimes audible, especially in the aortic area, and may be mistaken for the murmur of aortic regurgitation.‡

* *Specielle Diagnose der Innern Krankheiten*, vol. i. p. 14.

† *Text-book of Medicine*, English edition, p. 713.

‡ *Lehrbuch der Klinischen Untersuchungs-Methoden*, p. 282.

The systolic (hæmic or accidental) murmur in the mitral area is a source of great difficulty in diagnosis. The older observers, in general, denied its existence. Thus Walshe says, "The intra-cardiac hæmic murmur . . . is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and of the pulmonary artery . . . scarcely conducted in the direction of the aorta at all; frequently audible, on the contrary, at the second left, or pulmonary, cartilage; only in exceptional cases audible below the nipple; and never, within my experience, perceptible as far as the left apex, or outwards towards the axilla."* Walshe adds, "The doctrine of some observers that true blood-murmur may be systolic in time and seated at the apex appears to me wholly untenable; how deficiency of red particles in the blood can lead to its reflux through the mitral orifice I am at a loss to conceive."†

Sansom, on the other hand, found a hæmic murmur at the apex in 16 per cent. of his cases of anæmia, in 9 per cent. of which cases the apical murmur coexisted with a murmur in the pulmonary area, while in 7 per cent. it was present alone.‡

Clifford Allbutt writes, "There is no experience of the kind to which we may look back with more satisfaction than to systolic apex murmurs, which in their characters corresponded in all respects with those of permanent organic disease, but which disappeared entirely, nevertheless. Loud or harsh murmurs in this place are not so common, if I may speak for my own experience, as the softer murmurs; still, soft or harsh, they arise under like conditions of atony, and to our repeated

* Diseases of the Heart, fourth edition, p. 86.

† *Loc. cit.*

‡ Diagnosis of Diseases of the Heart, p. 273.

surprise clear . . . away altogether on appropriate treatment,"*

It is generally agreed that this murmur depends on temporary mitral regurgitation, due to muscular atony, usually in association with anæmia.

Great caution is, in my opinion, necessary in dealing with this murmur. The favourable type of case described by Allbutt is, fortunately, not rare. But I am also familiar with cases where the course of events is not so happy, where systolic apical murmurs persist in the case of young women, after the cure of their anæmia. Leube does not speak too strongly when he says, "*Die Unterscheidung dieser beiden Arten von Mitral-Insufficienz von einander ist unter allen Umständen sehr schwierig; die Differentialdiagnose übersteigt dabei nicht das Niveau der Wahrscheinlichkeit.*"†

There is a type of case which is fairly common, and in which an opinion may be given with some confidence, viz. where in presence of well-marked anæmia there is a loud systolic murmur in the pulmonary area, a faint systolic murmur in the mitral area, no or very slight dilatation of the left ventricle, and no history of rheumatism. That the mitral murmur in such cases is usually due to muscular atony depending on anæmia is, I think, certain, and it may be expected to pass away as the patient improves. But where the murmur is confined to the mitral area it is very difficult to be sure. Sansom says that a systolic murmur arising independently of structural disease "seldom attains its maximum audibility at the exact apex, but slightly to the right and left of it. It is usually soft, and does not replace the first sound. It does not occupy the whole, but the middle of the systole (it is

* System of Medicine, vol. v. p. 507, Art. Chlorosis.

† *Op. cit.*, vol. i. p. 15.

'meso-systolic'). It is much influenced by respiration ; it is intensified both during expiration and inspiration (especially the latter), but it often becomes inaudible at the end of the expiration." *

Broadbent says, "Hæmic murmurs are usually soft and blowing, not harsh or musical, and they do not replace or extinguish the first sound. Generally speaking, they are not conducted to the axilla or heard over the back, and are not accompanied by much displacement of the apex-beat. . . . Furthermore, it is the hæmic murmur which is most frequently late-systolic in time, that is, it follows the first sound at an appreciable interval instead of commencing synchronously with it." †

The above rules will, no doubt, sometimes prove helpful, and may at least suggest, if they do not establish, a correct diagnosis.

I doubt if there are any physical characters which can be positively relied upon to distinguish a systolic mitral murmur depending on muscular atony from a similar murmur depending on organic change in the valve.

In these difficult cases our chief reliance should, in my judgment, be placed on the following considerations :—

- (a) The history and constitutional state.
- (b) The condition of the left ventricle and the position and character of the cardiac impulse.
- (c) The site of maximum intensity of the murmur, its conduction, the variations in its characters depending on posture and movement.
- (d) The results of treatment.

A history of rheumatism weighs potently in the direction of a diagnosis of organic disease. The presence

* Allbutt's System of Medicine, vol. v., Art. Diseases of the Mitral Valve.

† Heart Disease, third edition, p. 189.

of well-marked anæmia suggests the probability that a murmur may be "hæmic" in character but in no way renders organic disease improbable. Functional or hæmic murmurs are, in my experience, most common in anæmic or under-nourished girls, who have exhausted their small store of strength either by hard work or by cycling, lawn tennis, or other such amusements.

As regards the condition of the left ventricle, any recognizable dilatation is unusual in anæmia, but may occur where overstrain is added to anæmia. A certain degree of fatty degeneration is found in anæmia. Definite hypertrophy may be taken as practically certain evidence of organic, as against functional, causation of a murmur.

The results of treatment are sometimes decisive, and it may be well in certain cases to wait for the light thus thrown upon our cases.

The diagnosis of "accidental" murmurs must often be by exclusion.

The systolic murmur of cardio-pulmonary origin.

This murmur may usually be recognized by the following characters: it is soft and superficial, variable from hour to hour or day to day, much influenced by the respiratory act and by posture. It may disappear when the breath is held. It is usually much louder in the recumbent than in the erect position.

Sansom thinks that the point of maximum intensity of this murmur is not at the exact apex, but a little to the right or the left of this point.

Potain taught that this murmur is "meso-systolic."

The most trustworthy criterion of this murmur is its close relation to respiration.

The systolic murmur due to pressure on a vessel by an enlarged gland or tumour.

Usually the locality of this murmur, and the general symptoms and history will suggest the correct diagnosis.

The same remarks might be made as regards the systolic murmur so often heard over the sac of an aneurism.

Diastolic Murmurs.

Diastolic murmurs may be audible either in the aortic area, or anywhere in the neighbourhood of the sternum.

They are rare in the mitral area.

The diastolic murmur of aortic regurgitation.

This important murmur has usually the following characters :—

It is usually low-pitched and long-drawn ; often blowing, sometimes ringing or definitely musical in quality ; rarely harsh or grating ; sometimes very faint and easily missed. Its point of maximum of intensity is variable. Its conduction is usually wide. It is not much under the influence of posture and movement. It is usually a genuinely diastolic murmur, *i.e.* it occurs with the second sound and is prolonged into the long pause. It may, however, occupy any portion of the diastole, even its concluding portion, so that the simulation of a mitral pre-systolic murmur is occasionally close. In such cases it is never, I believe, followed by the sudden "snap," *i.e.* the short, sharp, high-pitched first sound so characteristic of mitral obstruction.

In the majority of cases the diastolic murmur is preceded by a systolic murmur, which does not usually indicate aortic stenosis.

Diastolic murmur in the mitral area.

In the great majority of cases a diastolic murmur in the mitral area is a conducted aortic murmur.

A genuine diastolic mitral murmur is rare. It is, however, sometimes found in mitral obstruction in place of the much more usual pre-systolic murmur.

The "late-systolic" murmur, already described, is not uncommon. Its significance has been already considered. Some of these "late-systolic" murmurs are probably hæmic in origin.

Diastolic murmur in the pulmonary area.

This is usually the conducted aortic murmur.

In a few rare cases (especially in association with malignant endocarditis) a diastolic murmur indicative of pulmonary regurgitation has been recognized. This lesion may be distinguished from aortic regurgitation by observing that in pulmonary regurgitation the second sound is pure in the aortic area, and that the pulse does not possess the Corrigan quality.

Diastolic murmur heard in cases of extreme anæmia.

For a discussion of this point, see *ante*, p. 308.

Pre-systolic Murmurs.

A pre-systolic murmur may be present in the following conditions :—

- (a) Mitral obstruction.
- (b) Tricuspid obstruction.
- (c) Aortic regurgitation.

(a) *The pre-systolic murmur of mitral obstruction.*

This murmur is usually rough, rolling, vibratory, snoring, crescendo, or "ingravescent" in quality.

Its point of maximum intensity is at, or a little inside, the apex. Its conduction is in most cases for a short distance towards the sternum, exceptionally it is heard over a wide area.

The time is usually pre-systolic, *i.e.* it occurs just at the

end of diastole, and before the systole of the ventricles. In a few cases the murmur is genuinely diastolic, *i.e.* it occupies the greater part of the diastole.

Leube is of opinion that in certain cases there is a diastolic element and a pre-systolic element, separated by a distinct pause.* Fräntzel, also, holds this view. In a considerable number of cases this murmur is accompanied by a faint systolic murmur, indicative of slight regurgitation. The murmur is terminated by a sudden "snap," which is the short, sharp, high-pitched first sound.

I adhere to the view that the murmur is produced by the contraction of the hypertrophied and dilated left auricle.

This murmur is absent in a considerable number of cases of mitral obstruction.

(b) *The pre-systolic murmur of tricuspid obstruction.*

This is a very rare murmur, usually associated with the murmur of mitral obstruction, from which it is differentiated with difficulty.

Its quality resembles that of the murmur of mitral obstruction. Its site of maximum intensity is in the tricuspid area. It is accompanied by distension of the jugular veins, with little or no pulsation. Dropsy is usually prominent. Broadbent is of opinion that when dropsy appears early in a case of mitral obstruction, we may justifiably suspect the coexistence of tricuspid obstruction.†

(c) *The pre-systolic murmur in the mitral area, depending on aortic regurgitation (Flint's murmur).*

This is a comparatively common murmur, and may give rise to difficulties in diagnosis.

In a considerable number of cases of aortic regurgitation

* *Specielle Diagnose der Innern Krankheiten*, vol. i. p. 27.

† *Heart Disease*, p. 221.

a murmur is heard in the mitral area simulating the murmur of mitral obstruction.

Two views are held as regards its mechanism—

- (a) That it is due to the regurgitant current from the aorta impinging on the anterior flap of the mitral valve and throwing it into vibration; and
- (b) That the regurgitant current of blood floats up this flap of the mitral valve, prevents it from falling back, and so causes a relative narrowing of the mitral orifice.

This murmur, in my experience, does not usually present the characters of the murmur of mitral obstruction in their typical form. It is often a confused rumble, rather than a vibratory ingravescient murmur, and the typical "snap" is never present.

Attention to the sounds in the upper sternal region and to the pulse will usually suffice to prevent error.

(e) *Influence of posture and movement.*

This is an important matter, to which hardly sufficient attention is usually given. Posture and movement affect many murmurs, functional and organic, to a considerable degree; no general rules will cover all cases, and the facts require to be cautiously interpreted. Potain's dictum that "if the change from the dorsal decubitus to the sitting position causes the complete or almost complete disappearance of the murmur, it can be confidently ascribed to extra-cardiac causes" must be received with caution. Usually, however, we are not concerned with the total disappearance of a murmur on change of posture, which is upon the whole unusual, but with variations in the intensity of murmurs. It is not difficult to understand that posture and movement influence the blood pressure and the blood currents, and hence the quality and intensity of murmurs.

The following rules may be laid down as approximately correct regarding the influence of posture and movement upon cardiac murmurs, but exceptions will be found to occur.

- (a) The functional murmur in the pulmonary area is much influenced by posture, and is usually much more distinct in the recumbent position.
- (b) The systolic murmur in the mitral area, whether of functional or organic causation, is in some, but not all, cases influenced by posture, and is usually louder in the recumbent position. The influence of posture is more marked, as a rule, in the murmur due to anæmic or cardio-pulmonary causes than in that due to structural change in the mitral valve.
- (c) The pre-systolic murmur of mitral obstruction is not much influenced by posture, but is of all murmurs the most influenced by exercise and movement.
- (d) The systolic murmur in the aortic area, whether functional or organic, is more distinct in the standing position.
- (e) The diastolic murmur in the aortic area is not much influenced by posture.

LECTURE XVI.

PHYSICAL EXAMINATION OF THE BLOOD- VESSELS.

SUMMARY.

The Aorta—physical signs of aortic aneurism, diagnosis of atheroma of the aorta, seats of aortic aneurism.

Examination of the carotid, subclavian, axillary, brachial, and retinal arteries.

The Pulse—method of examining, the sphygmograph, characters of the pulse, viz.—

(a) Frequency.

(b) Volume.

(c) Tension.

(d) Rhythm.

Causes of increased frequency of the pulse.

Causes of diminished frequency of the pulse.

Alterations in the volume of the pulse.

Causes of high tension and of low tension, diastolic.

Varieties of arrhythmia.

The Veins—the jugular veins, turgidity of, movements in the veins, undulatory movements, pulsation.

Causes of venous pulse.

Venous hum.

Venous thrill.

The Capillaries—capillary pulse.

I SHALL devote the present lecture to the physical examination of the blood-vessels, in so far as that subject concerns the physician.

We shall consider—

A. The aorta, including its main branches.

B. The peripheral arteries. The pulse.

C. The veins.

D. The capillaries.

A. The Aorta.

The aorta may be investigated by the usual methods, viz., inspection, palpation, percussion, and auscultation. Inspection may reveal pulsation in various areas, palpation may confirm and extend our observation of such pulsation, percussion may reveal dulness, and auscultation the presence of murmurs.

Instead of pursuing the investigation of the aorta by these methods in the systematic manner which we have found convenient in the case of the heart, it will be simpler and more helpful to confine ourselves to the consideration of the affections of the aorta which are of clinical importance, viz.—

A. Acute aortitis.

B. Atheroma.

C. Aneurism.

(a) *Acute aortitis.*

Of this affection our knowledge is scanty. According to Broadbent, it occurs in the course of the acute infective diseases, and has been found associated with pregnancy, renal disease, fatigue, and rheumatism. The aorta is dilated, and its inner surface is irregular, uneven, and pinkish in colour for a distance of one and a half to two inches from the aortic valves. The symptoms consist of anginoid attacks attended by severe dyspnœa and a feeling of constriction in the region of the base of the heart. The pulse is rapid and sudden, vomiting may be present, and in some cases the temperature is raised. There are no characteristic physical signs, but there is evidence that the aorta is dilated.

The prognosis is unfavourable, and sudden death in an anginoid attack is the rule.*

* I am indebted for the above facts to Broadbent's account of this rare condition, viz. *Heart Disease*, pp. 362 *et seq.*

(b) *Atheroma of the aorta.*

This very common condition cannot always be diagnosed with certainty. It may exist without obvious changes in the arteries which are directly open to observation, *e.g.* the radials.

There are no definite physical signs of atheroma of the aorta, but accentuation of the aortic second sound is generally present, and a slight degree of hypertrophy of the left ventricle is common.

Dilatation of the aorta is usually present, and may sometimes be recognized by an area of dulness at the upper right edge of the sternum, and by visible pulsation in this region. A systolic murmur over the aorta is often present.

Additional significance will attach to the above signs if the patient be at or after middle life, and is the subject of alcoholism, gout, or syphilis.

The above-mentioned dulness on percussion may be masked by co-existing emphysema."

As long as perfect compensation is present there will be no symptoms in atheroma of the aorta. With failing compensation the usual train of symptoms will be set up.

It may be suspected that atheroma involves the coronary arteries if the following conditions are present :—

1. A slow and irregular pulse.
2. Syncopal, "apoplectic" or "epileptiform" attacks.
3. Anginoid pain.

(c) *Aneurism of the aorta.*

The arch of the aorta is a favourite seat of aneurism. In its earlier stages an aneurism in this situation may be latent, but in the course of its growth important signs appear, viz.—

Inspection.—There may be only a slight visible pulsation,

or there may be a definite bulging of the chest. This pulsation or bulging is most readily appreciated if we inspect the chest obliquely, the light falling upon it from the front. In aneurism of the ascending portion of the arch the bulging or pulsation is usually to the right of the sternum, about the level of the second right intercostal space, but with the growth of the aneurism it may involve the infraclavicular region, the manubrium sterni, and the third and fourth right intercostal spaces.

In aneurism of the transverse portion of the arch the pulsation or bulging may be either to the right or to the left of the sternum or in the episternal notch.

In aneurism of the descending portion of the aorta, the bulging or pulsation first appears posteriorly between the vertebral border of the left scapula and the spine, generally between the third and the sixth rib.

The tissues over the aneurismal sac may be eroded and thinned.

Palpation.—The bimanual method should be practised, one hand being placed upon the front of the sternum, and the other upon the back. In typical cases we feel a slow, heaving, expansile pulsation over the sac of the aneurism, synchronous with the systole of the heart, followed by a diastolic shock. If the sac of the aneurism contains much clot, the impulse may be weak.

Percussion.—Percussion must be practised with caution. Dulness will be noted over the area of bulging or pulsation.

Auscultation.—A systolic murmur is often present over the sac of the aneurism, but it is frequently absent, and inasmuch as it is common in the conditions which simulate aneurisms—*e.g.* vascular tumours—it is a sign of little value in the differential diagnosis.

A diastolic murmur is not uncommon. It may be due

to aortic insufficiency or simply to the stream of blood flowing back into the sac of the aorta during diastole. The aortic second sound is usually ringing or accentuated—a sign of much value.

The heart is frequently displaced, with or without hypertrophy, as the result of pressure by thoracic aneurism. The displacement is downwards and usually to the left. In the large aneurisms of the descending aorta the displacement may be to the right.

Hypertrophy is not usually marked in cases of aneurism where the cardiac valves are unaffected.

Among the general signs of aneurism may be enumerated—

Delay or difference in strength of the pulse in the radials.

Paralysis of a vocal chord, usually the left, depending on pressure upon the recurrent laryngeal nerve.

Changes in the pupil, depending on pressure upon the sympathetic.

Œdema of the neck and arms, depending on pressure on the superior cava.

Stridor, depending on pressure on the trachea.

Signs and symptoms simulating phthisis of the left apex, depending on pressure by the sac upon a bronchus or directly upon the lung.

Dysphagia, depending on pressure upon the œsophagus.

Vomiting, depending on pressure upon the vagus.

Intercostal neuralgia, depending on pressure upon nerves.

Hæmoptysis.

Tracheal tugging—a valuable and trustworthy sign of thoracic aneurisms, especially those of the transverse portion of the arch.

Differences in the strength of the two radial pulses requires to be interpreted with caution, inasmuch as they may depend upon anatomical peculiarities.

The so-called "aneurismal phthisis" may usually be recognized by attention to the history, general physical signs, and sputum. Leube regards "asthmatic" symptoms excited by change of posture as suggestive of aneurism.*

Seat of the aneurism :

(a) *Ascending aorta.*

The physical signs are dulness to the right of the manubrium sterni about the level of the second intercostal space, pulsation in the first and second spaces, displacement of the heart downwards and to the left, delay of the pulse in the peripheral arteries, accentuation of the aortic second sound. In some cases signs of pressure upon the superior vena cava appear ; in others the sac presses upon the root of the right lung.

This form of aneurism has been called the "aneurism of physical signs."

(b) *Innominate artery.*

The pulsation or bulging in this variety of aneurism is usually about the upper end of the sternum, or a little to the right, and may extend into the neck. The head of the clavicle may be dislocated.

(c) *Arch of the aorta.*

The physical signs are variable and sometimes slight, while the symptoms of pressure upon left recurrent laryngeal nerve, trachea, œsophagus, sympathetic and lung, are often marked, hence this aneurism has been called the "aneurism of symptoms." In some cases pulsation is visible in the second or third left space close to the sternum or in the

* *Specielle Diagnose der Innern Krankheiten*, p. 77.

episternal notch, dulness or percussion over the manubrium sterni and to the left of it may be recognized. There may be inequality in the size of the arteries of the head and neck on one side of the body, pressure upon the left innominate vein may cause congestion and œdema of the left side of the head and neck, a systolic murmur may be present, and the aortic second sound is accentuated.*

(d) *Descending thoracic aorta.*

The signs of aneurism in this situation are pulsation in the back to the left of the spine, weakening of the crural pulse, symptoms of pressure on the vena azygos and upon the vertebrae, with gradual erosion of these, stenosis of the œsophagus and of the left bronchus.

Physical signs may be ill-marked, and in some cases the only symptom is pain in the back, due to pressure upon one of the spinal nerves or vertebræ. In such cases the simulation of spinal caries may be close.

The carotid arteries.

The pulse in these arteries is not usually visible, but in persons with thin necks and of nervous habit there may be well-marked pulsation apart from disease.

Marked carotid pulsation may be due to—

(a) Nervous excitement.

(b) Exophthalmic goitre.

(c) Aortic regurgitation.

In the two former conditions the collapse of the artery following systole is much less marked than in aortic regurgitation.

Incipient or larval Graves's disease is a frequent source of error. Acceleration of the pulse, throbbing arteries and

* According to Osler (Practice of Medicine, fourth edition, p. 778), the tumour in aneurism of the transverse portion of the arch presents to the right of the sternum much more often than to the left.

nervous erethism may long precede either exophthalmos or enlargement of the thyroid. If the possibility of this condition be borne in mind, diagnosis will not be difficult.

The throbbing carotids of aortic regurgitation are easily recognized, first, by the physical signs in the heart, and, secondly, by the fact that the visible pulsation is continued into the small arteries.

A systolic murmur is often heard over the carotids, and may be due to conduction from the aortic valves or to pressure of the stethoscope.

A diastolic murmur is sometimes heard over the carotids in cases of aortic regurgitation, which is not a conducted murmur, but is produced *in situ* by the backward flowing stream of blood in the vessel passing the part narrowed by pressure of the stethoscope.*

The subclavian arteries.

These arteries may sometimes be seen to pulsate in health in thin persons. They may pulsate to a marked degree in aortic regurgitation.

A systolic murmur is often heard over these vessels, of which the significance is doubtful. It is a somewhat inconstant sign, and is probably not important.

Axillary and brachial arteries.

In aortic regurgitation visible pulsation in these arteries is intensified by raising the arm from the side.

In cases of advanced atheroma of these vessels, visible pulsation may be well marked, but the diastolic collapse, characteristic of aortic regurgitation, is not present. In atheroma the vessels are usually tortuous.

Retinal arteries.

Visible pulsation in these arteries is an important sign of aortic regurgitation.

* Gibson and Russell : Physical Diagnosis, third edition, p. 85.

B. The Radial Arteries—the Pulse.

The importance of a careful examination of the pulse and of the correct appreciation of the facts observed in connection with it, cannot be over-estimated. Both in diagnosis and in prognosis—more particularly, perhaps, in the latter—it is one of our chief guides. It gives information which is always of some, frequently of the highest, value.

In the examination of the pulse the patient should be seated or reclining, and the arm employed should be supported. It is convenient to examine the right pulse with the physician's right hand, and *vice versâ*. Two or three fingers should be employed, the index finger being on the side next the heart.

It is a good plan to examine the pulse at the beginning and again at the end of the interview, as in this way we get some measure of nervous aberrations. Both radials should be examined, and it is important to assure ourselves that there is no abnormal distribution of the vessels. Abnormalities in the distribution of the terminal arteries of the arm are not uncommon, and may lead to erroneous conclusions.

I shall not deal with the sphygmograph or sphygmographic tracings. The sphygmograph has thrown some interesting light on the physics of the circulation, but has given little genuine assistance in practical diagnosis.*

Before proceeding to investigate the characters of the pulse in detail, we should satisfy ourselves regarding the state of the arterial walls. The signs of atheroma are well known, and need not be described at length.

We examine the pulse in relation to the following characters :—

* For Broadbent's view on the value of the sphygmograph, see *The Pulse*, p. 32.

- (a) Frequency.
- (b) Volume.
- (c) Tension.
- (d) Rhythm.

a. Frequency.

The usual average in normal individuals is about 72 beats per minute; but the variations compatible with health are considerable. A pulse of 50 or 85 may be the norm for the individual.

The pulse is normally more frequent in women than in men, in childhood and youth and old age than in adult life, in small people than in tall persons. It is more frequent in the upright position, in the evenings, in very hot weather, during mental or physical exertion, during menstruation.

The pathological causes of increased frequency of the pulse are very numerous. The following causes may be mentioned.

1. Pyrexial conditions in general. Cerebral inflammations may be an exception. The degree of increased frequency of the pulse gives us some measure of the intensity of the toxæmia—*e.g.* in typhoid fever—but much depends upon age and temperament. A steady increase of the frequency of the pulse in fevers is of evil omen. This increase sometimes occurs while the other symptoms—*e.g.* pyrexia—remain stationary. In such cases cardiac failure may occur later. The exceptional frequency of the pulse in the early days of scarlatina, when a pulse rate of 120 to 150 is not uncommon, is of much diagnostic, but little prognostic importance. As a broad rule, the pulse is a better prognostic guide in fevers than the temperature. The case of scarlatina is exceptional.

2. Nervous diseases—*e.g.* exophthalmic goitre, functional palpitation, tachycardia, the early stage of meningitis, the late stage of cerebral tumour, tumour on the vagus.

3. Toxic conditions—*e.g.* chronic alcoholism, the excessive use of tea, coffee, or tobacco, large doses of digitalis and belladonna.

Persistent frequency of the pulse, apart from other obvious explanation, sometimes gives a valuable clue to concealed alcoholism and to incipient phthisis.

4. Organic heart disease. The frequency of the pulse is increased in all conditions attended by dilatation. It is usually increased in mitral obstruction, mitral regurgitation, and aortic regurgitation, but may be normal so long as compensation is well maintained. In aortic obstruction the frequency of the pulse is sometimes less than normal. In the senile heart the frequency of the pulse is usually increased, but may be diminished in certain cases of fatty degeneration, myocarditis, and coronary disease.

The term "tachycardia" is, I think, best reserved for those rare and interesting cases of extreme paroxysmal hurry of the heart, where the beats suddenly increase from 70 or 80 to 180 or 200, and then, after a time, return with equal abruptness to the normal.*

In myocarditis the pulse is, as a rule, small, frequent, and irregular.

According to Huchard, a marked increase of the frequency of the pulse on changes of posture and movement is very characteristic of arterio-sclerosis.†

The frequency of the pulse may be diminished in the following conditions :—

1. In convalescence from acute disease—*e.g.* pneumonia.

* See Lecture XVIII.

† *Traité Clinique des Maladies du Cœur et de l'Aorte*, vol. i. p. 330.

2. In many forms of brain disease—*e.g.* meningitis, tumor cerebri, apoplexy, abscess of the brain.

3. In certain cases of myocardial disease, aortic obstruction, arterio-sclerosis, atheroma of the coronary arteries.

5. In certain general diseases of the nervous system—*e.g.* melancholia, general paralysis, epilepsy, the gastric crises of tabes dorsalis.

6. In gastric diseases—*e.g.* chronic dyspepsia, carcinoma ventriculi, gastric ulcer.

7. In the course of treatment with digitalis and strophanthus.

Bradycardia is a vague term. It cannot conveniently be used as the converse of tachycardia, since the only variety of tachycardia which can be erected into a type is the paroxysmal variety, and, so far as I am aware, there is no such condition as paroxysmal bradycardia. To say that a patient has an abnormally slow pulse is to state a fact of much importance, but one to which no definite significance can be attached. We may, however, in a loose way speak of bradycardia, just as we speak of hyperpyrexia to indicate an abnormally high temperature, without implying any theory of its cause.

Slowing of the pulse must always be interpreted cautiously, but may give us suggestions of much value. Thus slowing of the pulse in connection with persistent headache, vomiting, and pyrexia is highly suggestive of meningitis. The connection of slow pulse with epilepsy is a fact of much importance. In some of these cases cerebral anæmia seems to be the proximate cause of the convulsive attacks.

In many cases of slow pulse, only every alternate beat of the heart reaches the wrist.*

* See Huchard, *op. cit.*, vol. i. p. 341.

(b) *Volume of the pulse.*

We distinguish between the volume of the pulse-wave and the volume of the blood-stream between the beats. Thus in aortic regurgitation the pulse-wave is commonly large in volume, while between the beats the vessel is comparatively empty. Normally, the volume of the pulse is increased during inspiration and diminished during expiration.

The volume of the pulse varies much in health.

The volume of the pulse is increased in many conditions, attended by low arterial tension, *e.g.* aortic regurgitation, febrile states.

The volume of the pulse is diminished in many cases of mitral disease, in aortic obstruction, and in states of inanition and collapse.

As a rule, when the tension of the pulse is high, the volume is small ; when the tension of the pulse is low, the volume is either large or small.

(c) *Tension of the pulse.*

Allbutt objects to the term "tension," which, he truly says, refers properly to the state of the vessel-walls, and not of the blood-stream. He prefers the term "tone." * The word "tension" is, however, generally employed to describe the blood-pressure in the vessels. It depends upon the tone of the arteries and the strength of systole.

The tension of the pulse is one of its most important characters. It may be approximately measured by the amount of force necessary to obliterate the pulse-wave between the beats. In states of high tension the artery remains full between the beats, and can sometimes be rolled beneath the finger. In states of low tension the vessel feels thready or even imperceptible between the beats.

* System of Medicine, vol. v. pp, 810, 811.

The high-tension pulse has to be distinguished from the sensation produced by a thickened artery. As a rule, the distinction is not difficult. The elasticity of the healthy vessel, the firm rise of the pulse-wave under the finger, the absence of irregularities on the surface of the artery, the amount of pressure necessary to obliterate the blood-stream are very characteristic of high tension, apart from recognizable disease of the vessels. It must be borne in mind that high-tension and arterio-sclerosis are very frequently conjoined, the former being the most frequent cause of the latter.

A low-tension pulse is often "dicrotic," *i.e.* after the primary beat there is a secondary beat ("a sort of echo of the main beat," as Broadbent calls it), due to the elastic recoil of the aorta. An extreme degree of dicrotism is sometimes spoken of as hyper-dicrotism.

Causes of high arterial tension.

The following causes may be enumerated :—

1. Luxurious living, free use of alcohol, sedentary habits, constipation.
2. Renal disease, especially granular kidney and acute and chronic desquamative nephritis.
The pulse-tension is not raised in pyelitis or in tuberculosis of the kidney.
3. Gout and glycosuria.
4. Plumbism.
5. Anæmia.
6. Aortic stenosis.
7. Pregnancy.
8. Raynaud's disease.
9. Emphysema.
10. Athleticism.

The full discussion of the above causes of high arterial

tension would take us far beyond the limits of these lectures. A few remarks must suffice.

Luxurious living, the free use of alcohol, sedentary life, and constipation probably act by producing a toxic condition of the blood, which in some way not quite clear leads to increased resistance in the capillaries.

The relation of renal disease to high arterial tension is close, but its precise rationale has not been fully made out. It is a good clinical rule to think of the kidneys when we find high arterial tension. Granular kidney is the typical case where high tension is usually well marked. In the so-called "small white kidney," also, the tension may be high,* and in certain cases of acute and chronic nephritis.

The high-tension pulse of gout, glycosuria and plumbism, is probably only a particular case of toxæmic high tension.

The high-tension pulse of anæmia and chlorosis, which is common but by no means invariable, has long attracted attention, inasmuch as it seems somewhat anomalous. In reality, however, the explanation is simple. The blood in anæmia is hydræmic, larger in quantity than usual, but deficient in corpuscular elements. An interesting confirmation of this view is the fact, to which my attention was first drawn by my colleague, Dr. Lorrain Smith, that the great majority of cases of well-marked chlorosis lose weight when put upon a course of iron, regulated diet, and rest. This loss of weight is easily explicable as due to the draining off of fluid from the blood and tissues. It is usually coincident with improvement in the patient's condition.

High arterial tension in aortic obstruction is due to the hypertrophy of the left ventricle which is usually present.

* See an interesting lecture by Dr. J. R. Bradford: Bright's Disease from a Clinical Standpoint.—*Lancet*, July 4, 1903.

Most observers are of opinion that high arterial tension is found, also, in certain cases of mitral obstruction. This has not been my experience. With arterial tension, which might be described as "fairly good" in mitral obstruction, I am familiar. Of genuine "high tension" in this disease I have no experience. The difference is, of course, only one of degree.

It seems doubtful whether the high arterial tension of pregnancy is due to "a general augmentation of the volume of the blood, or to the presence in the blood of effete matters derived from the foetus."*

The high arterial tension often present in Raynaud's disease is probably dependent on arterial spasm.

The high arterial tension sometimes observed in emphysema has been generally attributed to deficient oxygenation.

The high arterial tension apparently brought about by systematic severe physical exertion is important in these days of athleticism. It is a somewhat difficult matter to appraise correctly. That high arterial tension is frequent in professional athletes, and that it leads ultimately in a not inconsiderable number of cases to disease of heart, blood-vessels, or kidneys, is, I think, certain. The difficulty in such cases is to distinguish between the effects of physical exercise *per se* and the various concomitants—dietetic and otherwise—of the life of the average athlete. Healthy blood-vessels will stand with impunity an enormous amount of strain if the individual is young, careful in his habits regarding eating and drinking, and is free from rheumatism, gout, and syphilis.

Causes of low arterial tension.

Low arterial tension is found in the following conditions :—

* W. H. Broadbent : *The Pulse*, p. 159.

1. In most states of debility, malnutrition, and neurasthenia.

In anæmia, as already stated, the pulse-tension is often raised.

2. After food, hot drinks, a warm bath, fatigue, exhaustion.
3. In nearly all pyrexial conditions. Peritonitis is often an exception.

At the beginning of pyrexial conditions there may be a transient rise of tension.

4. In certain valvular affections of the heart, especially mitral regurgitation, the later stages of mitral stenosis and aortic regurgitation.
5. In myocarditis and fatty heart.
6. In obesity.

A persistently low arterial tension may be a constitutional peculiarity, and if the normal arterial tension of the individual is unknown to us we must be careful regarding the conclusions which we draw from low arterial tension.

(d) Rhythm of the pulse.

See Lecture XVIII., "Disorders of the Cardiac Rhythm."

C. The Veins.

The veins of the neck often give us the most important indications, but our survey should include the venous system generally.

The veins of the neck.

In health the external jugular is usually visible, the internal jugular usually invisible. The external jugular may be invisible in stout people, especially in the erect position.

The veins are overfull in the following conditions :—

1. Violent expiratory efforts, as in obstruction of the trachea larynx or bronchi, whooping-cough, capillary bronchitis.
2. Chronic bronchial catarrh and emphysema.
3. Dilatation of the right auricle.
4. Compression or thrombosis of the inferior cava, the innominate vein or the jugulars. In many such cases the distension is unilateral.
5. Conditions where increase of intrathoracic pressure is present, *e.g.* large pericardial and pleural effusions, large mediastinal tumours, aneurisms of the aorta.

In advanced emphysema the swelling of the cervical veins is very marked (*a*) because of the feeble action of the dilated right auricle, and (*b*) because, owing to the atrophy of the pulmonary capillaries, the amount of blood passing through the lungs is much reduced.

The swelling of the veins of the neck is much influenced by posture, being more evident in the recumbent than in the sitting or standing position, because in the recumbent position the flow of blood into the auricle is retarded. The respiratory movements, also, have much influence, the cervical veins tending to collapse during inspiration and to become distended during expiration. According to Guttmann there is one case where this law is reversed. "In rare cases, particularly of fibrous mediastinitis, the reverse of these appearances may be seen, distension of the jugular veins with each inspiration: the fibrous cords developed in this affection exercise a certain amount of traction on the intrathoracic veins each time the chest is expanded in inspiration, and in that way lessen the lumen of the vessel in question." *

* P. Guttmann: Handbook of Physical Diagnosis, p. 226. See also Sahli: Lehrbuch der Klinischen Untersuchungs-Methoden, p. 134.

Turgidity of the cervical veins is usually increased in a marked degree by coughing.

Movements in the veins.

The movements in the veins are of two kinds.

(a) Undulatory movements, depending on respiration.

(b) Pulsation, depending on a reflux current from the right heart (venous pulse).

The undulatory movements dependent on respiration consist of a gradual filling of the veins during forced expiration and a sudden emptying of the veins at the beginning of inspiration. "The explanation is obvious: the fulness during forced expiration is the result of the positive pressure on the veins of the thorax during that phase, which causes a retrograde wave of blood to close the valve above the jugular bulb; the sudden emptying attending inspiration is produced by the negative pressure within the thorax at that time, which draws the blood onward in the direction of the heart." *

The two common conditions in which this undulatory movement in the cervical veins is most marked are advanced pulmonary emphysema and mitral obstruction.

The movements are undulatory because the veins, once thrown into commotion, do not immediately return to a state of quiescence.

Venous pulse.

Visible pulsation may in certain cases be present in the veins in health. This physiological pulsation may be distinguished from the pathological pulsation, presently to be considered, by observing that on pressing with the finger on the vein pulsation on the side next the heart is either obliterated or rendered weaker. The contrary is

* Gibson and Russell: Physical Diagnosis, third edition, p. 100.

the case in pulsation dependent on a reflux current of blood from the right auricle.*

The venous pulse is best observed in the external jugular vein and in the recumbent position. It is most easily appreciated in thin persons. The venous pulse consists either of a single or a double throb. Its time is either systolic or pre-systolic. It depends upon the regurgitation of a wave of blood from the right auricle through the vena cava superior, and the innominate veins to the jugulars. It is usually more marked in the right side, inasmuch as the right innominate vein is continuous in an almost direct line with the vena cava superior.

So long as the jugular valves continue competent, the pulsation is limited to the lower portion of the vessels, especially to the sinus, but beyond this point an undulation in the vein is perceptible, due to the shock conveyed to the valve by the recurrent stream of blood. Later, when the jugular valves give way, pulsation becomes clearly perceptible in the veins, and may rise as high as the angle of the jaw.

Venous pulsation in the neck is practically pathognomonic of tricuspid regurgitation.

The venous pulse may easily be distinguished from transmitted carotid pulsation by altering the position of the head, so that the arteries and the veins are no longer in close juxtaposition, and by observing the effect of pressure by the finger on the pulsation. If the pulsation be venous, it will be intensified on the side next the heart.

The diagnosis of tricuspid regurgitation may, in certain cases, be confirmed by grasping the liver between the two hands and satisfying ourselves that its volume is increased by the venous pulsations. It is not always easy to

* Sahli, *op. cit.*, p. 135.

distinguish hepatic pulsation from the impulse conveyed to the liver by an hypertrophied and dilated right ventricle.

Venous hum.

Lænnec thought this sound was produced in the arteries. It is now known to be produced in the jugular veins. It is due to vibrations of the venous walls.

This well-known sound has the following characters:—

(a) *Quality.*

It is a humming, roaring, sighing, or blowing sound, sometimes definitely musical, soft or harsh, weak or loud. Its intensity depends in part upon the rapidity of the current in the veins. It is increased by moderate pressure of the stethoscope, obliterated by heavy pressure.

In the great majority of cases the sound is continuous, but if it be exceptionally feeble we may only hear it at the moment of its greatest intensity, *e.g.* during diastole and during inspiration.

(b) *Point of maximum intensity.*

It is usually best heard between the two attachments of the sterno-mastoid muscle. It is often louder on the right side than on the left.

(c) *Line of conduction.*

It is conducted along the jugular veins. It is sometimes heard over the manubrium sterni, and even in the aortic area.

(d) *Time.*

It is a continuous murmur, but it is louder during diastole and during inspiration.

(e) *Effect of posture.*

It is usually much louder in the sitting than in the recumbent position, and may even disappear when the patient lies down. It is sometimes better heard when the patient turns his head to the opposite side.

Accompanying the venous hum a thrill can often be felt above the clavicle.

A feeble venous hum is not uncommon in health, and has no pathological significance. The loud hum is practically pathognomonic of anæmia.

A venous hum is sometimes heard over the femoral veins.

In exophthalmic goitre a venous hum may be audible over the thyroid.

D. The Capillaries.

Normally, no pulsation is visible in the capillaries. A capillary pulse may, however, be observed in certain cases attended by hyperæmia, or in other cases (*e.g.* aortic regurgitation, Graves's disease, chlorosis, fever) where there is a sudden pulse wave.

A capillary pulse may be observed sometimes in the nails, where an alternate flushing and pallor may be seen, or we may evoke the phenomenon by drawing the nail across the forehead and producing a red line, where a similar alternation may be preceptible.

Capillary pulsation is very characteristic of aortic regurgitation. It is most evident during the stage of compensation. It is often absent.

LECTURE XVII.

THE STUDY OF CARDIAC SYMPTOMS.

SUMMARY.

- A. Symptoms referrible to the Heart itself,**
Viz. Palpitation—functional and organic; pain—character, seat, radiation, response to remedies, angina and pseudo-angina.
- B. Symptoms referrible to the Blood-vessels,**
viz. Abnormal throbbings, irritable aorta.
- C. Symptoms referrible to the Integumentary System,**
viz. Pallor, cyanosis, œdema.
- D. Symptoms referrible to the Respiratory System,**
viz. Dyspnœa, “cardiac asthma,” orthopnœa, Cheyne-Stokes respiration, cough, hæmoptysis, yawning, hiccough.
- E. Symptoms referrible to the Digestive System,**
viz. Dyspepsia, pain, vomiting.
- F. Symptoms referrible to the Urinary System,**
viz. Alterations in the amount, colour, and specific gravity of the urine, presence of albumen, casts, and blood.
- G. Symptoms referrible to the Nervous System,**
viz. Pain, syncope, vertigo, headache, insomnia, impaired intellectual power.

HEART disease affects in some degree all the viscera, hence the symptoms of cardiac disorder are multiform.*

The following classification of the symptoms of heart disease will be found practically convenient:—

A. Symptoms referrible to the heart itself.

* The line between signs and symptoms cannot be rigidly drawn. The former are, in the main, objective, *i.e.* observed by the physician. The latter are, in the main, subjective, *i.e.* felt by the patient. But the distinction is not really a logical one. Cough, for example, is both a sign and a symptom. I use the term “symptoms” in this chapter in the popular sense as a matter of practical convenience.

- B. Symptoms referrible to the blood-vessels.
- C. Symptoms referrible to the integumentary system.
- D. Symptoms referrible to the respiratory system.
- E. Symptoms referrible to the digestive system.
- F. Symptoms referrible to the urinary system.
- G. Symptoms referrible to the nervous system.

This arrangement involves to some extent the inconvenience of a cross-division. Thus cardiac pain might be considered either under A or G. Nevertheless, this classification of symptoms is easy to apply in practice, and tends to completeness. If the symptoms of heart disease are investigated under these various heads, nothing important will be overlooked.

A. Symptoms referrible to the Heart itself.

Of these we may enumerate the following :—

- (1) Palpitation.
- (2) Pain, including feelings of weight and oppression.
- (3) Tenderness.

(1) Palpitation.

By this term we understand the subjective sense of painful or excessive movements of the heart. The movements are often felt to be violent or irregular. Sometimes the sensation is that of "sinking," or of suspension of the heart's action. It is to be noted that palpitation has no necessary connection with the actual movements of the heart. These may be violent or irregular, while the patient remains entirely unconscious of them, while, on the other hand, patients may complain of palpitation when to the observer the heart's action seems normal. The essential fact in palpitation is increased sensitiveness of the sensorium, rendering it morbidly conscious of the heart's action.

Palpitation is common in both functional and organic

diseases of the heart. Many of the most violent forms of palpitation are toxic in origin—due to tea, tobacco, or alcohol, or are due to emotion—fright, grief, anxiety, or depend upon dyspepsia, or are part and parcel of a nervous heredity. These forms of palpitation are often accompanied by murmurs, and the differential diagnosis from palpitation of organic causation is not always easy.

Functional palpitation may generally be recognized by attention to the following points:—

- (a) The patient is neurotic or dyspeptic, probably of the female sex and under fifty years of age. Exceptions to the age rule are, however, not uncommon.
- (b) The subjective feeling of distress is marked, and the palpitation is loudly complained of.
- (c) The heart is not appreciably altered in its dimensions.
- (d) Some definite cause for the palpitation can often be assigned, *e.g.* dyspepsia, nervous heredity, abuse of tea, tobacco, or alcohol, emotion.
- (e) The first sound is short and loud in the mitral area, or the two sounds may be approximately similar in quality. If murmurs are present, they will be systolic in time, and in most cases will have their maximum in the pulmonary area.
- (f) The pulse tension is low.
- (g) Dropsy, congestion of the lungs, cyanosis, hæmoptysis, and albuminuria are absent.
- (h) Carminatives and sedatives relieve the palpitation, which is also favourably influenced by fresh air, moderate exercise, and taking a few deep breaths.

Whether functional palpitation ever causes appreciable hypertrophy of the heart is a moot point. I have never seen a case where this seemed certain.

Palpitation of organic causation may usually be recognized by attention to the following points:—

- (a) The patient is probably rheumatic, or the subject of vascular degeneration.
- (b) The subjective feeling of distress is less than in functional palpitation, except in very advanced cases, where the diagnosis is obvious.
- (c) The heart is usually more or less hypertrophied or dilated.
- (d) The palpitation is usually brought on, or at least aggravated, by exertion.
- (e) Posture has some, often much, influence. The patient usually feels easiest in the sitting position.
- (f) The pulse tension is usually low, but may be high.
- (g) The sounds of the heart may present many variations, and murmurs—systolic, pre-systolic, or diastolic—may be present.
- (h) Dropsy, congestion of the lungs, cyanosis, hæmoptysis, and albuminuria may or may not be present.
- (i) Cardiac tonics relieve.

(2) *Pain.*

Cardiac pain is common in both functional and organic heart disease. Pain is, on the whole, commoner in functional than in organic conditions. In organic disease, pain is relatively common in aortic disease, rare in mitral disease.

Angina pectoris stands by itself, as the precise nature and mechanism of the pain are not known.

We may consider cardiac pain under the following heads:—

Character.

The pain of angina pectoris—agonizing, vice-like, paroxysmal—is well known.

The pain of aneurism is constant, with exacerbations,

of variable but usually considerable intensity, gnawing, boring, or lancinating in character.

The pain of aortic disease is variable, often takes the form of præcordial oppression, may be anginoid.

The pain of functional heart disease is often much complained of, may be dull and aching, or sharp and stabbing. Exaggerated sensitiveness is usually present.

Seat.

Cardiac pain is usually described as superficial and on the left side. Its seat corresponds to the distribution of the spinal nerves from the eighth cervical to the sixth dorsal.

In some few cases the pain is bilateral.

The usual limitation of cardiac pain to the left side is related to the fact that the left ventricle and the aorta are subjected to much higher pressure than the right ventricle and pulmonary artery. The pain is often particularly severe at the apex, or in the third left space.* The pain is superficial, and is never referred to the heart directly.

Radiation.

The pain of true angina usually radiates into the left arm and hand, sometimes into the left neck, exceptionally to the right side, and even into the loins and legs.

In aortic disease, radiation into the left arm is common.

In functional disorders radiation is indefinite, but the patient is often neurotic, and may complain of pains in many parts of the body, *e.g.* head, back, ovarian region.

Radiation of pain has usually some relation to its intensity. The more intense the pain, the wider the area of radiation, and *vice versa*.

* W. H. Broadbent : Heart Disease, p. 352.

If the pain be due to pressure upon the cardiac nerves by an aneurism or a neoplasm, the area of radiation will be determined by anatomical considerations.

Response to treatment.

The pain of angina usually yields to the nitrites. The pain of aortic disease, which is sometimes anginoid in character and associated with coronary disease, is often relieved by iodide of potash, belladonna, and bromides. The nitrites are sometimes useful in aortic disease. Aconite is better avoided. The pain of functional disorder is relieved by aperients, carminatives, or tonics. Bromides should be used sparingly, and alcohol is better avoided. If gout or rheumatism be present, the treatment appropriate to these conditions will relieve cardiac pain.

The term pseudo-angina has been applied to various forms of cardiac pain which simulate angina pectoris. On the whole, it is a misleading term; it suggests an affinity of functional heart disorder with angina pectoris which does not exist.

(3) *Tenderness.*

Tenderness on pressure often accompanies cardiac pain. It is sometimes particularly marked at the edge of the left mamma and over the second left rib.*

The interpretation of cardiac pain and tenderness opens up a wide field of inquiry. Cardiac pain usually means much to the patient, but it may mean much or little to the physician. Angina pectoris, aortic valvular disease, and aneurism should be inquired for, and will often be recognized without much difficulty. In the young, the neurotic, and the dyspeptic, the probability that cardiac pain is of functional origin is always strong. Cardiac

* Broadbent, *op. cit.*, p. 353.

pain should be estimated cautiously where we have certain evidence of vascular degeneration, as in such cases coronary disease is not improbable. The clue of causation is often of much value. In a doubtful case the temporary prohibition of tea, tobacco, or alcohol may clear the case up. Pain brought on by exertion or accompanied by dyspnœa must always be viewed with suspicion.

Marked tenderness over the præcordial area has been in my experience much commoner in functional than in organic cases.

B. Symptoms referrible to the Blood-vessels.

Patients often complain of abnormal throbbings in various vessels, rushing sounds in the ears, and the like.

The most important of these, inasmuch as it is rather common and may lead to difficulty in diagnosis, is the condition known as "irritable aorta." In this condition the abdominal aorta throbs violently, the patient complains of much pain and distress, and the suspicion of aneurism may arise.

The differential diagnosis may usually be determined by attention to the following points:—

- (a) The patient is usually a woman, and more or less neurotic.
- (b) The aorta can be felt to beat violently in the direction of its length, but no saccular distension can be made out.
- (c) The pain is often described as severe, but it is variable in character and in seat, and does not resemble the persistent boring pain of aneurism. It is not usually present in the back.
- (d) There are none of the indirect signs of aneurism,

- e.g.* changes in the pupil, diminution of the pulse in the femoral artery.
- (*e*) There is usually no evidence of vascular degeneration.
- (*f*) A murmur may be present over the aorta in these cases. It is systolic in time, as in the case of aneurism, and has no weight in determining the diagnosis.

C. Symptoms referrible to the Integumentary System.

Of these the most important are—

1. Pallor.
2. Cyanosis.
3. Œdema.

1. *Pallor.*

Pallor is common in functional heart disorder, where anæmia is often a factor.

It is also common in aortic disease.

It is not a common feature in mitral disease.

2. *Cyanosis.*

Cyanosis is a marked feature in many cases of congenital heart disease. It is present in an extreme degree in tricuspid regurgitation. It is found in both forms of mitral disease, when compensation has begun to fail, but is, on the whole, more characteristic of mitral obstruction than of mitral regurgitation. Some observers believe that it is present in slight degree even in well-compensated mitral disease. It is not a common feature of aortic disease, where pallor is more usual, but arises when the mitral valve becomes secondarily involved. It is unusual in fatty heart and in aneurism. It is never present in functional heart disease.

Cyanosis is increased by coughing, stooping, and by

intercurrent pulmonary affections. It is usually most marked in the face, especially the lips, but may be general. It is sometimes easily observed in the fingernails.

3. *Œdema.*

Œdema is a leading feature of cardiac disease. Impeded venous return is the usual determining cause.

A moderate degree of œdema of the feet and ankles is common in anæmia, especially in chlorotic girls whose occupation compels them to stand for many hours continuously. This form of œdema often disappears after rest and sleep.

Cardiac œdema has the following characters:—

It begins inferiorly, in the lower extremities, but in advanced cases becomes general, and includes the face. It is aggravated by fatigue and by pulmonary complications. Its most common cause is mitral regurgitation. It is less common in mitral obstruction. As in both these conditions dilatation of the right ventricle and tricuspid regurgitation may be present, it is not clear why œdema should be commoner in the former lesion than in the latter. Broadbent suggests that the explanation may be that in mitral obstruction the diminished output from the left ventricle does not allow of sufficient pressure in the capillaries to give rise to effusion of serum.*

Œdema does not occur in aortic disease so long as the mitral valve remains competent, but it is common in the later stages of aortic regurgitation, when the mitral valve becomes implicated.

Œdema is rare in myocarditis and fatty heart.

Cardiac œdema is often accompanied by passive

* Heart Disease, p. 70.

effusion into the serous cavities—most often the pleura, frequently the peritoneum, rarely the pericardium. It might have been supposed that passive effusion would follow some obvious mechanical law, *e.g.* that if present in one pleural cavity, it would also be present in the other. This is not the case. It is common to find a large effusion into one pleural cavity, while no fluid can be discovered in the other, but it is probable that in many of these cases there is a slight effusion on the side apparently unaffected.

Nor is there any fixed law as to the correlation of the different forms of passive effusion and œdema. I have seen a case of mitral obstruction where there was a large ascitic collection, for which the patient sought admission to hospital, but no hydrothorax and no œdema of the extremities.

These facts suggest that some other factor than mechanical laws is at work in such cases.

This factor may have relation to the state of nutrition of the vessels.

D. Symptoms referrible to the Respiratory System.

These are among the most constant features of cardiac disease, and are of great importance.

We may enumerate the following :—

1. Dyspnœa, including the so-called “cardiac asthma,” orthopnœa, Cheyne-Stokes respiration.
2. Cough.
3. Hæmoptysis.
4. Yawning and hiccough.

1. Dyspnœa.

Dyspnœa is practically a constant feature of all forms of organic heart disease where compensation has begun to fail.

The immediate causes of dyspnœa are the following :—

- (a) Faulty supply of blood to the nerve-centres.
- (b) Passive congestion and œdema of the lungs.
- (c) Hydrothorax.
- (d) Passive congestion of the stomach, liver, and bowels, interfering with the movements of the diaphragm.

Cardiac dyspnœa may assume the following forms :—

- (a) A tendency to sigh, apart from emotional cause.
- (b) Breathlessness on exertion, usually felt at first on going upstairs or ascending an incline, later manifesting itself on walking upon the level, and eventually arising in connection with any form of effort.
- (c) Breathlessness which is continuous, with exacerbations, not depending upon exertion, but much aggravated by it. In most of these cases pulmonary complications are present.
- (d) Orthopnœa, *i.e.* the condition in which the patient is unable to lie down. Insomnia is usually marked in these cases.
- (e) Cheyne-Stokes respiration, *i.e.* a form of dyspnœa in which the breathing consists of cycles, each beginning with complete apnœa, the respirations gradually increasing in depth and frequency until dyspnœa has reached its maximum, and then again declining until apnœa is once more established.
- (f) A feeling of præcordial oppression, the patient being afraid to breathe, or finding the respiratory act positively painful.

The relation of dyspnœa of organic heart disease to exertion is close, but we must bear in mind that exertion will also excite or aggravate the dyspnœa of anæmia, emphysema, and renal disease.

Orthopnœa is very suggestive of organic heart disease. Cheyne-Stokes respiration suggests lesions of the myocardium, but has many other causes.

In the investigation of cardiac dyspnœa we must bear in mind the causes of dyspnœa which are independent of organic heart disease. The chief of these are the following :—

- (a) Anæmia.
- (b) Emphysema, asthma.
- (c) Acute febrile disease.
- (d) Laryngitis and laryngeal obstruction.
- (e) Renal disease.
- (f) Hysteria, bulbar paralysis, lesions of the vagus nerve.

Most of these conditions can be recognized or excluded without much difficulty.

The dyspnœa of anæmia is often marked, and is frequently much aggravated by exertion. It does not take the form of orthopnœa, is unaccompanied by cyanosis or by effusion into serous cavities, and the pulse tension is often raised.

The dyspnœa of emphysema and of asthma is characterized by great prolongation of the expiration, which is not a feature of cardiac dyspnœa. The history and physical signs in the chest will usually make these cases clear.

Renal disease sometimes causes a form of dyspnœa closely resembling asthma. The history and the state of the urine are the best guides in such cases.

Laryngeal dyspnœa is characterized by hoarseness, stridor, cough, perhaps cyanosis. Blood may be hawked up.

The dyspnœa of hysteria is often very characteristic. The chief point is the extreme frequency of the respirations,

which may number sixty or seventy per minute, without much actual distress, and without cyanosis or notable alteration of the pulse. Sometimes the patient sputters at the mouth, and is obviously hysterical.

The Cheyne-Stokes form of dyspnœa calls for a more detailed description.

In this remarkable form of dyspnœa we have a series of respirations of gradually increasing force and accelerating rate until a maximum is reached. To these succeed a descending series of respirations of gradually diminishing force and amplitude until breathing is brought to a standstill (apnœa). After an interval the cycle begins again with a fresh crescendo series. The patient is usually conscious during the period of dyspnœa, unconscious during apnœa. During apnœa the pupillary reflex is usually abolished. Cheyne-Stokes respiration occurs in association with arterio-sclerosis, aortic regurgitation, uræmia, fatty heart, apoplexy, and sunstroke.

It is always a most grave symptom, but does not always portend an immediately fatal issue.

2. *Cough.*

The cough of cardiac disease presents nothing distinctive.

It may depend upon bronchial catarrh, passive congestion of the lungs, œdema of the lungs, pulmonary infarction, hydrothorax.

It is usually accompanied by more or less expectoration, which in some cases is tinged with blood.

3. *Hæmoptysis.*

Hæmoptysis is a common feature of heart disease. Its immediate cause is usually passive congestion of the lungs, depending on mitral disease, more often upon

mitral obstruction than upon mitral regurgitation. It is rare in aortic disease. It is a usual feature in pulmonary infarction. It is frequent in thoracic aneurism.

As regards the amount and character of the blood no general rules can be laid down.

It should be borne in mind that mitral disease and tubercle, the two commonest causes of hæmoptysis, are not often present in the same subject.

4. *Yawning and hiccough* are sometimes troublesome features of cardiac disease.

E. Symptoms referrible to the Digestive System.

Dyspepsia plays a large part in connection with cardiac disease, partly as effect, partly as aggravating concomitant. On the one hand, cardiac disease may set up chronic gastritis with its train of symptoms, while on the other hand dilatation of the stomach or flatulent distension of that organ may seriously embarrass the enfeebled heart and be a source of serious danger. The old saw, "When the patient complains of his heart, think of his stomach ; when he complains of his stomach, think of his heart," should not be forgotten.

In gastritis from cardiac disease there is usually the following train of symptoms—furred tongue, nausea, impaired appetite, discomfort after food, pain on pressure in the epigastric region, irregular bowels, sometimes melæna. Vomiting may be present. Jaundice is not uncommon.

Dyspepsia may be one of the earliest symptoms of failing compensation in cardiac disease, and may prevail throughout the course of the case. It is often aggravated by an unsuitable dietary mainly composed of carbohydrates.

F. Symptoms referrible to the Urinary System.

The urine in cardiac disease may be normal. Usually it is reduced in amount, the quantity of pigment is increased, the specific gravity is raised (1025–1030), albumen and casts may be present. Lithates are often abundant.

The amount of albumen in the urine in cardiac disease varies greatly. It may be only a trace or very abundant. The casts present are generally few in number. Hyaline casts are common; granular and blood casts somewhat rare.

When albuminuria is present in the urine and a cardiac lesion exists, it may be an important question whether the albuminuria depends on the cardiac lesion or upon an independent and co-existing nephritis.

The chief guides in this connection are as follows:—

- (a) The nature of the casts present. Hyaline, granular, or blood casts may be present either in passive congestion of the kidneys from cardiac disease or in nephritis. Fatty, epithelial or lardaceous casts are strong evidence of independent kidney disease.
- (b) The amount of urea excreted. The amount of urea excreted is diminished in all forms of chronic Bright's disease, often to an important extent. It is not usually much altered in cardiac disease.

In connection with the association of cardiac and renal disease, Dickinson writes as follows: "In advanced cases of renal disease, where the cardio-vascular changes are conspicuous and the heart greatly enlarged, it is probable that the regurgitation is brought about by the disease of the kidney; but more often when cardiac and renal symptoms present themselves together, the cardiac is primary, the renal secondary. . . . On the whole, the

kidneys are tolerant of the congestion of cardiac disease. Persistent change is slow to establish itself. The urine, after being albuminous, may, under treatment, cease to be so. Uræmia seldom declares itself. Dropsy, when present, is more cardiac than renal." *

G. Symptoms referrible to the Nervous System.

Of these we may enumerate the following:—

Pain.

Syncope.

Vertigo.

Visual and auditory phenomena.

Headache.

Insomnia.

Impaired intellectual power.

Cardiac pain has been already considered.

Syncope.

Syncopal attacks are amongst the common phenomena of cardiac disease. Their immediate cause is probably failure of the circulation in the nerve-centres. They may be of all grades of severity, from a transient faintishness to a fatal syncope.

Syncopal attacks may occur in any form of cardiac disease attended by failure of compensation. They are common in the later stages of both mitral and aortic disease, in myocarditis and fatty heart, and sometimes occur in pericardial effusion, angina pectoris, aneurism, and thrombosis of the pulmonary artery. Syncope is not uncommon in functional heart disorder, as in anæmia, fatigue, emotion. Syncope in aortic disease is often accompanied by pain. Syncope in mitral disease is often accompanied by cyanosis and dropsy.

* Allbutt : System of Medicine, vol. iv. p. 379.

Syncope in connection with fatty heart may come on without warning and prove fatal.

Syncope is often precipitated by exertion, emotion, a full meal, or reflex irritation, *e.g.* from the uterus, the urinary organs, the bowels, or the skin.

Vertigo.

Vertigo is a common feature of cardiac disease.

It may occur in any form of cardiac disease, functional or organic, valvular or myocardial. It is more characteristic of aortic than of mitral disease. It is very common in myocardial affections. The chief point of importance in this connection is to distinguish vertigo of cardiac origin from vertigo due to dyspepsia, neurosis, Menière's disease, or renal disorder.

The vertigo of Menière's disease is characterized by its intensity, its paroxysmal character, and the presence of deafness, sometimes of vomiting.

The possibility of *petit mal* should be remembered.

The vertigo of cardiac disease is usually accompanied by dyspnoea, palpitation, and the physical signs of organic disease.

Abuse of tobacco is a common cause of vertigo.

Various visual and auditory phenomena, e.g. muscæ volitantes, flashes of light before the eyes, booming noises in the ears, may be present in cardiac disease.

Headache is occasionally present in cardiac disease.

Insomnia is often one of the leading features of cardiac disease, and may be one of the chief sources of the patient's sufferings.

It is usually marked in cases where orthopnoea is present. It may take the form of simple wakefulness, or a tendency to drop off into an uneasy slumber, and then wake up with a start, hideous dreams alternating with inability to sleep.

Insomnia may alternate with a soporose condition. It is common to find cardiac patients drowsy and longing to sleep, but unable to do so, or waking up after a brief and unrefreshing slumber.

Impaired intellectual power.

This is common in cardiac disease, and most often takes the form of indisposition to mental effort and readiness to become fatigued after slight intellectual exertion.

LECTURE XVIII.

DISORDERS OF THE CARDIAC RHYTHM.

SUMMARY.

Frequency of cardiac arrhythmia, its variable import, difficulty of classifying the various forms of arrhythmia, co-existing conditions which affect the significance of arrhythmia.

Varieties of arrhythmia—

Simple intermission.

Simple irregularity.

The coupled rhythm (*Rythme couplé du cœur*).

Reduplication of the cardiac sounds.

Gallop-rhythm.

Embryocardia.

Tremor cordis.

Delirium cordis.

Tachycardia.

Bradycardia.

Causes, general characteristics and prognostic indications of the above varieties of arrhythmia.

DISORDERS of the cardiac rhythm are common in both functional and organic heart disease. They may be of trivial or of grave import. They may be the index of transient nervous disturbance, of a toxæmia, or of important changes in the valves or the myocardium. Their interpretation is often difficult, their bearing on prognosis and treatment obscure. They are, therefore, the more deserving of careful study.

There is an arrhythmia of the heart and an arrhythmia of the pulse. In most, perhaps all, cases of intermittent pulse, the intermission is coincident with an ineffectual systole, which may be recognized by auscultation.

The classification of the various forms of cardiac arrhythmia is not very satisfactory.

An ætiological classification would be the best. We might endeavour to distinguish the following groups:—

- (a) Arrhythmia of nervous origin—where the nervous mechanisms of the heart are at fault. To this group belongs the large class of cases where toxic causes—tea, tobacco, alcohol—are present.
- (b) Arrhythmia of mechanical origin—where the physics of the circulation are disturbed, as in mitral disease.
- (c) Arrhythmia of myocardial origin—where the myocardium is primarily at fault, as in fatty degeneration of the heart.

This classification is not, however, practically convenient, because it is often impossible to distinguish these three groups; and because some forms of arrhythmia belong to more than one group. The line between (b) and (c) can hardly be drawn.

We are therefore compelled to classify the varieties of arrhythmia according to their more obvious physical characters, *e.g.* intermittency, simple irregularity, gallop-rhythm, and the like. It is well to recognize that the terms in use are somewhat loose and do not necessarily correspond to definite pathological states. The following classification is the one usually adopted:—

Varieties of Cardiac Arrhythmia.

- (a) Simple intermission.
- (b) Simple irregularity.
- (c) The coupled rhythm.
- (d) Reduplication of the cardiac sounds.
- (e) Gallop-rhythm.
- (f) Embryocardia.
- (g) Tremor cordis.

(h) Delirium cordis.

(i) Tachycardia.

(j) Bradycardia.

(a) *Simple intermission.*

In this condition a beat at the pulse is "missed" or "dropped," usually at fixed intervals—one in five, ten, fifteen, or twenty, as the case may be—the heart's action being otherwise regular. On listening over the heart at the moment of intermission, we can usually satisfy ourselves that the "missed" beat at the radials is coincident with an incomplete and abortive systole of the ventricles.

The missed beat is often followed by a "bounce," which is "the leap of the heart against the low pressure of the unfilled arteries." *

According to Balfour, intermission is "a reflex inhibition of the heart through the vagus." † This may be the true explanation of intermission present in toxic states and in heart disease, but this theory is not easy to reconcile with the well-known fact that intermission is occasionally physiological.

As already mentioned, the intermission is usually more or less rhythmical, *i.e.* it occurs at fixed intervals. This rhythm is, however, easily disturbed and modified by intercurrent causes, *e.g.* pyrexial attacks, nervous excitement, and the like. Under such influences the intermission of the heart may be temporarily suspended.

The causes of intermission are as follows:—

1. Toxic causes, *e.g.* tea, tobacco, digitalis, gout, dyspepsia. This group of causes is by far the largest.
2. Nervous causes, *e.g.* shock, nervous debility, hypochondriasis.

* Allbutt : System of Medicine, vol. v. p. 816.

† The Senile Heart, p. 43.

3. Cardiac causes, *e.g.* aortic disease, fatty heart, strain of the heart.

4. Febrile causes, *e.g.* pneumonia, typhoid fever.

As already mentioned, intermission is sometimes normal and physiological. Intermission may be conscious or unconscious. Huchard thinks the latter the more grave.* The significance of intermission depends absolutely upon cause and the nature of co-existing lesions, if such be present. In itself, intermission is a symptom not admitting of any definite interpretation.

Many of the causes above enumerated will be sufficiently evident—*e.g.* pneumonia, typhoid fever, aortic disease. In the absence of other obvious explanation, the assumption that intermission is due to some toxic cause will not often be found erroneous, but we shall require to assure ourselves, so far as may be possible, that neither valvular disease nor fatty heart is present. A careful inquiry should be made regarding the use of tea, coffee, tobacco, alcohol, and drugs. Nor should we be easily satisfied that because there is no manifest and gross excess in the use of these articles their operation can be excluded. In such cases, we have no measure of individual susceptibility. A special quality of tea, a particular brand of cigars, may excite intermission, while the patient may be able to make use of other varieties of these articles without inconvenience. In any particular case where the cause of intermission is obscure, it will not be amiss to make the experiment of the temporary withdrawal of some article of food or luxury known as a potential cause of this condition. Failing assistance from this quarter, we should look for dyspepsia, examine the teeth, inquire into dietetic habits, the sufficiency of mastication, the condition of the bowels. If these causes can be excluded, we should suspect some

* *Traité Clinique des Maladies du Cœur et de l'Aorte*, vol. i. p. 336.

neurosis, and make inquiry for causes which tend to debilitate the nervous system—*e.g.* worry, disappointment, loss of sleep, prolonged attendance upon the sick, and the like.

If toxic causes, dyspepsia, and nervous debility can be excluded, and no acute disease be present, intermission is either a personal peculiarity—possibly of no significance—or it depends upon organic cardiac changes, especially fatty degeneration and dilatation. As a broad rule, however, intermission is not sufficient ground for suspecting organic heart disease. Such disease will, if present, in most cases present its characteristic signs. Intermission has never in my experience occurred as an isolated symptom in organic heart disease, either valvular or myocardial.

Intermission is a very serious sign in aortic regurgitation, in pneumonia, and in typhoid fever.

(b) *Simple irregularity.*

By "simple" irregularity I mean irregularity without definite, distinguishing characteristic. It is evident that the forms of arrhythmia which remain to be considered—*e.g.* gallop-rhythm, embryocardia, tachycardia—are specialized forms of irregularity. We shall here consider irregularity which does not present any special peculiarity.

Irregularity usually involves an arrhythmia both of time, of tension, and of volume. The beats succeed each other at unequal units of time, the intervals between the beats may be shorter or longer without any fixed rule, several short intervals may be succeeded by one long interval, or the cardiac action may be apparently without method. The volume of the pulse, also, varies within considerable limits. Variable, also, is the tension, but

I think in less degree, as the tension in these cases is nearly always low.

An important question meets us here *in limine*. Does an irregular pulse ever occur in perfect health?

Allbutt says, "In my own experience I have often met with an irregular pulse in smokers—never, I think, in the normal state." *

On the other hand, Gibson says, "It is certainly true that the pulse, even in absolute health, may be very irregular;" and again, "In young persons it (*i.e.* simple irregularity) may simply be the result of the adaptation of the different functions of the body." †

My experience inclines me to the latter view. In a certain number of persons the pulse seems to me occasionally irregular, either in consequence of trifling nervous excitement or in relation with the respiratory function—in both instances consistently with perfect health. In most of these cases the irregularity is transient. I am not clear that I have ever met persistent irregularity under normal conditions.‡

Causes of irregularity of the heart.

1. Mitral disease—either obstructive or regurgitant.
2. Pericarditis.
3. Myocardial disease.
4. Toxic causes—tea, tobacco, alcohol, digitalis, gout.
5. Dyspepsia.
6. Anæmia.
7. Acute specific diseases—*e.g.* pneumonia, typhoid fever.
8. Brain diseases.

* System of Medicine, vol. v. p. 814.

† G. A. Gibson: Diseases of the Heart and the Aorta, pp. 188, 189.

‡ For an ingenious and elaborate, if not always convincing, account of the different forms of cardiac irregularity, see Huchard's work, vol. i. pp. 337 *et seq.*

9. Emotional shock.
10. Bronchitis and emphysema.

Mitral disease.

There is some conflict of opinion regarding irregularity of the pulse in mitral disease.

Sansom says, "Irregularity of the pulse is not, in my experience, a characteristic of mitral insufficiency," and again, "It is to be noted that a very marked irregularity of the heart's rhythm is by no means infrequent in mitral stenosis. . . . The irregularity of rhythm is evident to the auscultator. Such irregularities may be entirely due to disturbances of the nervous mechanism, and may be quite independent of structural changes in the heart; but when signs of organic valve disease coexist with it, mitral stenosis is the lesion in the great majority of cases."*

Strümpell says, "The pulse in mitral regurgitation is quite strong and usually regular." "The radial pulse is small in every case of severe mitral stenosis, and is very often irregular."†

Bramwell says, "The pulse in some cases (*i.e.* of mitral regurgitation) is quite regular; in others markedly irregular." "So long as the left auricle is able to empty itself (*i.e.* in mitral stenosis), the pulse may be of good volume, good tension, and perfectly regular, but it usually happens that in consequence of over-distension the muscular tissue of the left auricle is every now and again stimulated to premature contraction, which, passing to the muscular tissue of the ventricle, is manifested at the wrist in the form of an imperfect pulsation. . . . In the later periods of the case, and especially when the cavity of the left auricle is constantly over-distended, and

* Allbutt : System of Medicine, vol. v. pp. 983, 1021.

† A Text-book of Medicine, Eng. edition, pp. 313, 315.

its muscular fibre in a condition of irritable weakness, the pulse becomes quick and extremely irregular.” *

There is no doubt that so long as compensation is fully maintained irregularity of the pulse is unlikely to occur either in mitral obstruction or mitral regurgitation. It is equally certain that in advanced stages of broken compensation irregularity is common in both lesions. The point which remains open is—Upon the whole, is irregularity characteristic of mitral obstruction and not characteristic of mitral regurgitation? To this question I should be disposed to give an answer in the negative. Some observers are of opinion that irregularity may occur in mitral obstruction even in the stage of perfect compensation.

Huchard describes two forms of arrhythmia which occur in mitral regurgitation. “L’une appartient aux dernières périodes de la maladie, elle est contemporaine de l’asystole, elle résiste presque toujours à la digitale et comporte un pronostic grave : c’est une arythmie d’origine myocardique, puisqu’elle est liée à la dégénérescence du muscle cardiaque. L’autre peut se montrer à une période rapprochée du début, elle est d’origine mécanique, elle est souvent modifiée favorablement par la digitale et son pronostic est bénin.” †

Pericarditis is a cause of irregularity. It may be associated with myocardial disease.

In fatty heart, fibroid myocarditis, and other such conditions, the action of the heart is commonly irregular, but this is by no means invariable. In such cases irregularity is often accompanied by anginoid pain, breathlessness, and syncopal attacks, but these may all be absent.

* Diseases of the Heart, p. 492.

† *Traité Clinique des Maladies du Cœur et de l’Aorte*, vol. i. p. 349.

Toxic causes tend more often to produce intermission than irregularity, but they are also frequent causes of the latter condition.

The other causes of irregularity need not be dwelt upon in detail.

The importance of the cerebral causes is obviously great.

Irregularity is, upon the whole, more serious than intermission, inasmuch as it less often owns a toxic causation, and is more often associated with grave organic changes in the heart, *e.g.* dilatation or fatty degeneration. Nevertheless, irregularity of the heart's action may be compatible with long years of vigorous life, and its gravity depends less upon the symptom *per se* than upon its cause and the associated conditions. As a general rule, irregularity is less serious in the young, in whom nervous causes are most often operative, than in the old, where the defect is more likely to be in the circulatory apparatus. Irregularity brought on by exertion is more serious than irregularity which is favourably influenced by exertion.

Irregularity is of more or less grave significance if associated with any of the following conditions:—

Dropsy, anginoid pain, persistent breathlessness, pulmonary complications, cerebral disease, syncope, acute febrile diseases.

In the absence of such concomitants and of definite proof of structural disease of the heart, irregularity of the heart's action must not be too gravely regarded. In the remaining cases, toxic causes, usually removable, or neurotic causes or anæmia, admitting of cure or palliation, are commonly present.

(c) *The coupled rhythm (Rythme couplé du cœur).*

In this condition two beats of the heart occur with a short pause between the beats, and the second beat is

followed by a much longer pause, this cycle being then repeated. This is the *pulsus bigeminus*.

Sometimes, but rarely, the two beats are of equal force. Much more commonly the first beat is relatively strong, and the second beat relatively weak. It is excessively rare for this rule to be reversed, and the second beat to be the stronger of the two. In a considerable proportion of cases the second beat is too weak to reach the wrist, but is appreciable over the heart and in the carotids. A large proportion of cases of abnormally slow pulse are due to each alternate systole of the ventricles being too weak for the impulse to reach the wrist.

In some cases, the rhythm is not double, but triple or quadruple.

Huchard regards the coupled rhythm as indicative of degeneration of the myocardium and of the nervous supply of the heart. He thinks the types above enumerated represent lesions of varying degrees of gravity, the least serious being the condition where the two beats are of approximately equal force, the most serious where the second beat is too weak to reach the wrist.*

The coupled rhythm is also occasionally present in valvular disease of the heart, in anæmia, in typhoid fever, and in diseases of the bulb, and in pressure on the vagus nerve.†

(d) *Reduplication of the cardiac sounds.*

This is a difficult and obscure subject. I must own myself entirely sceptical regarding many of the current theories on the subject. Asynchronism either of the ventricles or of the semilunar valves seems to me on many grounds improbable. Nor do the theories of Potain, Sansom, and other leading authorities carry conviction.

* *Op. cit.*, vol. i. p. 341.

† *Ibid.*, p. 342.

It may not be amiss to recall the somewhat negative views of Walshe on this subject. "In regard of diagnostic significance it must be confessed these reduplications are almost valueless in the present state of knowledge. And for the following reasons: reduplication is never, as far as I have observed, permanent, and invariable; it occurs most commonly in hearts either healthy or temporarily disordered in function only; less commonly in cases of slight organic affection; and with least frequency when serious valvular disease exists; it is not connected, as a rule, with any particular form of disease, either of the heart itself, or of the system, rheumatic or other; it comes and goes in the course of a few beats of the heart; sometimes disappears on change of posture, and may be affected, nay induced, by the act of respiration."*

On this passage we may pass at least one criticism. The so-called reduplication of the second sound is without doubt, as Sansom has well insisted, closely related to mitral obstruction, and may be regarded as one of the signs of that condition. It has been suggested that in such cases one of the elements of the so-called reduplication is due to the contraction of the walls of the hypertrophied left auricle. It is an objection to this theory that "reduplication" is a fluctuating sign, and that it is often absent in cases of mitral obstruction.

(e) *Gallop-rhythm.*

This is the well-known cantering rhythm, where the sounds of the heart have a triple rhythm, with the accent usually on the last element.

Potain, who was the first to give careful attention to this sign, regarded it as due to doubling of the first sound,

* Diseases of the Heart, fourth edition, p. 75.

and thought that it was specially associated with granular kidneys.

According to Sahli the accent is upon the second element at the apex, upon the third element over the great vessels.* The same authority is of opinion that gallop-rhythm is distinguishable from the pre-systolic murmur of mitral obstruction chiefly by the fact that it is equally audible over a wide area of the præcordium.

A systolic and a diastolic gallop-rhythm, a left ventricle and a right ventricle gallop-rhythm, have been described.

Whatever be the explanation of this remarkable sign, the gallop-rhythm is important. It is found in dilated hearts with failing compensation, in granular kidneys, and in Graves's disease. My experience is in accord with that of Graham Steel, who writes as follows regarding this sign: "The true *bruit de galop* (*Der Galopprhythmus*) is a sign always best heard over the ventricles, *i.e.* over the chief part of the muscle of the heart, and though occasionally it may be heard widely distributed, and even audible at the base, in the latter situation it is less loud than over the body of the heart. The *bruit de galop* may be heard in all forms of muscle failure of the heart—even in that due to anæmia—but the most perfect examples are supplied by cases of chronic Bright's disease, when the vigour of the hypertrophied heart muscle is on the wane. . . . It means a failing heart muscle." †

(f) *Embryocardia.*

This is a condition first recognized by Stokes, in which the long pause is shortened, the first sound comes to resemble the second, and the cardiac sounds approximate to the tic-tac of the foetal heart.

This phenomenon indicates profound weakness of the

* *Op. cit.*, p. 258.

† *Encyclopædia Medica*, vol. iv. p. 425.

cardiac muscle, usually associated with dilatation, and is most often heard in the later stages of prolonged fevers or wasting diseases.

(g) *Tremor cordis.*

This phenomenon has been well described by Balfour.*

It is a species of "fluttering," or trembling of the heart. That organ "trembles like an aspen leaf." There is no violent throbbing as in palpitation, but simply a rapid, tremulous fluttering. The attacks occur without warning, and are often associated with flatulence or gastric disturbance. They may occur either in a heart structurally sound or they may accompany any form of organic disease. Emotion is not an exciting cause. The attacks last a few seconds only, and though very alarming to the patient, pass off harmlessly.

(h) *Delirium cordis.*

This term is applied to an extreme degree of cardiac arrhythmia, in which many forms of irregularity are combined. It occurs in dilated heart, especially in association with mitral obstruction. It is, on the whole, a grave condition, but is consistent with long years of life.

(i) *Tachycardia.*

If the term tachycardia is to serve any useful end in medicine, it must be limited to the well-known paroxysmal variety of excessively rapid heart action, what Bristowe calls "paroxysmal hurry of the heart." If we apply the term "tachycardia" to every case of extremely frequent cardiac action, we make the term meaningless, and hinder the elucidation of a remarkably interesting condition.

Tachycardia, thus defined, is a rare affection. Apart from Graves's disease, which is of course not uncommon, I have met with only two well-marked examples. As these

* G. W. Balfour : The Senile Heart, pp. 64 *et seq.*

cases are rare, it may be well to record the two which have fallen within my experience.

CASE I.—A. B., female, aged 35, hospital patient. She had enjoyed good health until a few weeks previous to admission to hospital. There was no history of rheumatism or of alcoholism.

The first attack occurred as a consequence of nervous shock, in connection with a street fight in which her husband was engaged. I saw her in many subsequent attacks. The heart beats would jump up in a moment from 75 or 80 to 180 or 190. On one or two occasions the attack came on when my finger was on the patient's pulse, and I was demonstrating the case to my clinical class. The attacks could not be attributed to any definite cause, they occurred at all hours, and under the most various conditions, lasting usually from twenty to thirty minutes, sometimes as long as four or five hours, in one case for an entire day. During the attack the patient was weak, pallid, and breathless, perspired freely, but remained quite conscious. The pulse was small in volume, low in tension, and fairly regular. Digitalis proved useless, but the patient always improved more or less under rest and a course of bromides and belladonna. Usually, after four or five weeks' treatment in hospital, the attacks diminished in frequency, and the patient decided to go home. She sought re-admission on several occasions, extending over a period of four or five years. Gradually, the amount of improvement derived from treatment grew less. No valvular defect could be made out, but there was marked visible pulsation in the carotids, and a certain amount of hypertrophy and dilatation began to be set up. The patient ultimately died in the Belfast Union Hospital, but I have no details as to the final stage of her illness. The duration of the case was about five or six years.

CASE II.—The patient is a gentleman, aged 43, and the attacks have occurred at variable intervals for twenty years. The pulse rate runs up from 75 or 80 to 140, 150, or even 180. The attacks last several hours, and come and go with absolute suddenness. The patient is able to go about during an attack, but feels rather weak after it is over. He has no actual dyspnœa.

The cause in this case is obscure. The patient has suffered from neuralgia of the brachial plexus. There is no family history of nervous disease. He is slightly rheumatic. He is not anæmic. He used to be a great tea-drinker, smokes very moderately, and is a teetotaler. He has had a good deal of business worry. He is rather dyspeptic. The heart is not hypertrophied or dilated. There is a systolic murmur heard generally over the præcordia, loud in the pulmonary area, weak at the apex, soon lost on listening towards the axilla. The heart's action is somewhat irregular. The tension of the pulse is good.

It is remarkable that in this case the attacks of tachycardia have lasted for twenty years without any decided failure of health. The patient is still fairly well, and actively engaged in business. His condition has, however, changed somewhat for the worse during the last year.

While the above are the only two typical cases of genuine paroxysmal tachycardia which have arisen in my practice, I have seen many cases of heart hurry in association with Graves's disease, in dilatation depending on valvular defect, in neurotic palpitation and other conditions. These latter cases, however, as owning a definite nosological relation, are best studied in connection with the diseases of which they form one of the manifestations, but "paroxysmal tachycardia" is a clinical entity, and is a useful term, if employed within strict limitations.

Tachycardia is probably a genuine neurosis, and has no definite relation to Graves's disease, hysteria, or organic heart disease. It has been attributed, without any satisfactory evidence, to paralysis of the vagus, and also to irritation of the sympathetic. Sahli inclines to the idea of an epileptoid discharge from the accelerator nerves of the heart.*

The prognosis in tachycardia is bad as regards cure, doubtful as regards duration. Cases often run a prolonged course; but the heart tends to get worn out, and become dilated. We know that this is not true, as a general rule, of mere nervous palpitation, which may last indefinitely without producing structural change in the heart, and which does not tend to shorten life. It would seem as if tachycardia represents a more profound disorganization of the innervation of the heart than functional palpitation.

(j) *Bradycardia.*

While tachycardia, strictly defined, may deserve to rank as a disease, *i.e.* it represents (like Graves's disease, epilepsy, and chorea) a fairly well-defined group of clinical symptoms which have some more or less constant characters, bradycardia is only a clinical label. It has no definite connotation, and simply informs us that in such and such a case an abnormally slow pulse is present. There is no "paroxysmal" bradycardia to correspond to "paroxysmal" tachycardia. Nevertheless, while bradycardia is not a disease, it is a condition worth studying. If we may talk without absurdity of hyperpyrexia, and group together the causes of that condition, and inquire into its significance, so may we do in the case of bradycardia. No inconvenience will arise if we do not erect bradycardia into a disease type.

* *Op. cit.*, p. 832.

The term bradycardia may be applied to any condition where the pulse is habitually, or for any considerable period, under 50.

It must be remembered that in a very large proportion of these cases there is an abortive and ineffectual systole of the ventricles alternating with each effective systole.

Bradycardia may be a constitutional peculiarity. It will be remembered that Corvisart records that Napoleon's pulse was 40 ; but he was epileptic, and the association of bradycardia with epilepsy is one of the most outstanding facts relating to this condition.

Bradycardia may occur in certain physiological states, especially after delivery, and as the result of hunger.

Bradycardia is, upon the whole, rare under 40 or 50.

The pathological causes of bradycardia are numerous, though the condition is not really very common.

We may enumerate the following :—

- (a) States of exhaustion—*e.g.* in the course of pneumonia, scarlatina, diphtheria, small-pox, typhoid fever, erysipelas ; after extreme fatigue or excessive hunger ; sexual excess and masturbation.
- (b) Disorders of the digestive system—*e.g.* dyspepsia, cancer of the stomach, dilatation of the stomach, gastric ulcer, jaundice.*
- (c) Certain toxic conditions—*e.g.* carbonic acid poisoning, uræmia, lead poisoning, poisoning by digitalis, tobacco, belladonna, aconite, colchicum, opium, veratrum viride.
- (d) Certain diseases of the nervous system—*e.g.* epilepsy, apoplexy, cerebral tumour, meningitis,

* For a very complete discussion of the relation of bradycardia and gastric disease, see Riegel in Nothnagel's System of Medicine, vol. Diseases of the Stomach, English edition, pp. 160 *et seq.*

bulbar disease, melancholia, hysteria, neurasthenia, sunstroke, injury to the cervical spine, compression of the vagi.

(e) Organic heart disease—*e.g.* myocarditis, fatty degeneration, aortic obstruction, mitral obstruction.

(f) Anæmia.

(g) Pain.

We may, with advantage, distinguish the following varieties of bradycardia :—

1. *The bradycardia of childhood.*

This is rare. We should inquire for—

(a) Digestive disorders—*e.g.* gastric catarrh, worms.

(b) Nervous disorders—*e.g.* neurasthenia (including the form depending on masturbation), cerebral tumour, meningitis, epilepsy, injury to spine.

(c) Anæmia and chlorosis.

(d) The specific fevers.

2. *The bradycardia of adult life.*

We should inquire for—

(a) Toxic causes—viz. tobacco, digitalis, lead, uræmia.

(b) Digestive causes—viz. dyspepsia, diseases of stomach.

(c) Nervous causes—viz. epilepsy, melancholia, cerebral tumour, meningitis, cerebral hæmorrhage, injury to cord.

(d) Circulatory causes—viz. aortic obstruction, mitral obstruction.

3. *The bradycardia of old age.*

We should inquire for—

(a) Myocarditis, fatty degeneration of heart, aortic obstruction.

- (b) Toxic causes — viz. tobacco, digitalis, lead, uræmia.
- (c) Nervous causes—viz. epilepsy, melancholia, cerebral tumour, meningitis, cerebral hæmorrhage, injury to cord.

Bradycardia must be interpreted with caution. It may mean much or little. The discovery of the cause is essential to its rational interpretation.

Where bradycardia stands alone, and there is no disease of myocardium, cardiac valves, kidneys, or nervous system, it is safe to affirm that its import is not usually grave.

LECTURE XIX.

SOME DIAGNOSTIC PROBLEMS IN CONNECTION WITH HEART DISEASE.

SUMMARY.

- Significance of a systolic murmur having its maximum intensity at the aortic cartilage or the adjacent portion of the sternum.
- Significance of a pre-systolic murmur audible along the left edge of the sternum and in the mitral area.
- Significance of a diastolic murmur audible in the mitral area.
- Significance of a systolic murmur audible over the whole præcordial area.
- Significance of a systolic murmur audible in the mitral area.
- Significance of a systolic murmur audible at and to right of apex.
- Significance of a systolic murmur arising in the mitral area in the course of chorea.
- Possibility of mitral regurgitation disappearing.
- Tricuspid murmurs.
- Diagnosis of myocardial changes.

I PROPOSE in the present lecture to consider a few of the diagnostic problems which present themselves in connection with cardiac disease.

A. A systolic murmur is audible, having its maximum intensity at the aortic cartilage or the adjacent portion of the sternum.

What are the possible explanations?

- (a) Aortic obstruction.
- (b) Dilatation of the aorta, acute aortitis, roughening of the intima of the aorta.
- (c) Aneurism.
- (d) Anæmia.

The differentiation of the above causes is not always possible. Definite aortic obstruction being undoubtedly a very rare lesion, the presumption is always against its presence, unless the state of the left ventricle and of the pulse decisively confirms the diagnosis. Moderate hypertrophy of the left ventricle, a slow pulse of moderate volume and rather high tension, a rough, harsh murmur with its maximum intensity at or about the aortic cartilage, partial or complete inaudibility of the aortic second sound, the male sex and middle life,—such a combination of signs is typical of aortic obstruction. Such cases have occurred with extreme rarity in my experience.

At a late stage of aortic obstruction with failing left ventricle the murmur may become soft and weak.

Dilatation of the arch of the aorta is a common condition, which may sometimes be made out by careful percussion. Acute aortitis cannot be certainly recognized by our present methods of diagnosis.

It is questioned whether mere roughening of the aortic valves or of the intima of the aorta will produce a murmur in the absence of constriction or incompetence. Probably this is not a common cause of murmurs, but it is quite conceivable that an extreme degree of roughening might cause an eddy in the blood-current, hence a vibration of the walls of the aorta, and so give rise to a murmur.

Aneurism may give rise to the murmur in question. We may expect to establish its existence by careful inspection and percussion, by attention to the history and symptoms, and by attending to tracheal tugging, the state of the radial pulse, and the state of the pupils. Aortic aneurism is usually accompanied by a ringing, accentuated, aortic second sound, which may serve to differentiate the condition from aortic obstruction, where this sound is weak or inaudible.

The murmur of anæmia very rarely has its maximum in the aortic area, though it is not very rare to find it audible there. In the great majority of such cases it will be found to be louder in the pulmonary area; it is not well conducted, it is much influenced by posture, and the presence of a venous hum may assist the diagnosis.

The state of the pulse may assist us in analyzing the murmur in question. The somewhat small, hard, regular, and rather infrequent pulse of aortic obstruction is characteristic and important. The full, regular, fairly strong, and moderately frequent pulse of anæmia must be noted. These characters are, however, by no means constant in anæmia.

Inequality in the two radial pulses is common in aortic aneurism.

The presence of a systolic thrill at the base may be found either in aortic obstruction or in aneurism. Diastolic shock is characteristic of aneurism.

The conduction of a systolic murmur heard with maximum intensity at or near the aortic cartilage may throw important light upon its nature. The murmur of aortic obstruction is conducted well into the large vessels of the neck, also sometimes to the apex. The murmur of dilatation of the aorta or of roughening of the aortic valves or of the intima of the aorta is conducted along the sub-clavians and the carotids. The murmur of anæmia is not well conducted in any direction. The murmur of aneurism is not conducted much beyond the area of the aneurismal sac.

B. A pre-systolic murmur is audible along the left edge of the sternum and in the mitral area.

How shall we determine whether this murmur is due to mitral obstruction or to a recurrent stream of blood from incompetent aortic valves, *i.e.* a Flint's murmur?

As a rule, this question can be answered without difficulty. Attention should be paid to the following considerations :—

- (a) Flint's murmur rarely possesses the rough, vibratory, "ingravescent" quality, typical of mitral obstruction.
- (b) Flint's murmur is comparatively variable and inconstant.
- (c) Flint's murmur is not followed by the short, sharp, high-pitched first sound, characteristic of mitral obstruction.
- (d) Flint's murmur is accompanied by the usual physical signs of aortic regurgitation, viz. a diastolic murmur audible over a wide area in the neighbourhood of the sternum, hypertrophy of the left ventricle, Corrigan's pulse.
- (e) In mitral obstruction the left ventricle is not appreciably hypertrophied, except in those cases where regurgitation has preceded or accompanies the obstruction, epigastric pulsation is common, cyanosis is frequent, the pulse is small in volume, often irregular, not collapsing, tension variable but often pretty good.
- (f) The throbbing arteries of aortic regurgitation are often pathognomonic and quite unlike the vascular conditions of mitral disease.

C. A diastolic murmur is audible in the mitral area.

How shall we determine whether it is a conducted aortic murmur or one of those exceptional cases where the murmur of mitral obstruction is diastolic, rather than pre-systolic, in time?

The line of reasoning available in the preceding case (B), is for the most part applicable here.

An examination of the base of the heart will usually establish the fact of aortic regurgitation, if this lesion be present.

The diastolic murmur of aortic regurgitation is not uncommonly audible at the apex, but in such cases it will almost invariably have its point of maximum intensity either at midsternum or along the left or the right edge of the sternum. The diastolic murmur sometimes present in mitral obstruction is not usually heard beyond the bounds of the mitral area. The conduction of the aortic diastolic murmur is more extensive than that of the mitral diastolic murmur.

The condition of the left ventricle, the state of the arteries of the neck, the characters of the pulse, already sufficiently adverted to, may give important indications.

A history of hæmoptysis or of embolism favours mitral obstruction rather than aortic regurgitation.

It should be borne in mind that a considerable number of cases, diagnosed during life as cases of mitral obstruction, have been shown at the autopsy to be cases of aortic regurgitation.

D. A systolic murmur is audible over the whole præcordial area.

How shall we determine its seat and origin?

In this case we have usually to deal with one of the following conditions :—

(a) Congenital heart disease—possibly pulmonary obstruction, with imperfection of the inter-auricular or inter-ventricular septa.

In such cases the youth of the patient, the history of symptoms from birth, the presence and degree of cyanosis, the clubbed fingers, general chilliness and imperfect nutrition, make the diagnosis easy.

- (*b*) An exceptionally loud and well-conducted murmur of mitral regurgitation.

In this case the murmur will have its maximum intensity at the apex, it will be well conducted into the axilla, and probably to the left back, it will be faintest at the aortic cartilage, the left ventricle will be hypertrophied and dilated, the pulse will have the characters of mitral regurgitation.

- (*c*) A combination of mitral regurgitation with dilatation of the aorta, roughening of the aortic valves or the intima of the aorta, or (less probably) with aortic obstruction.

In this case there will probably be two areas of intensity of the murmurs; one in the aortic region and the other at the apex. There may be a middle region where the murmurs are both faint.

The condition of the left ventricle and of the pulse will be such as we find in mitral regurgitation.

- (*d*) A combination of mitral regurgitation with functional murmur at the base.

This is not uncommon.

According to Balfour, Naunyn, Leube, and others, the murmur of mitral regurgitation has in certain cases its maximum intensity in the second left intercostal space.

This is a doubtful doctrine.

In many cases with a systolic murmur in the mitral area and all the typical signs of mitral regurgitation, and also a systolic murmur in the second left space, it may be difficult to say whether the latter murmur is conducted from the mitral orifice or is hæmic in character. From the practical point of view, this question is not one of much moment.

It must be borne in mind that a systolic murmur in the pulmonary area is by no means confined to cases of overt anæmia. It is common in mere debility, and not rare in states of apparently perfect health.

E. A systolic murmur is heard in the mitral area in an anæmic patient.

How shall we determine whether the murmur is the sign of permanent organic defect of the mitral valve, or depends upon temporary dilatation of the left ventricle, the result of muscular weakness consequent on anæmia?

This is a most important question, which cannot always be answered with certainty. We have already considered it in some of its phases.*

The question is not whether in the case supposed mitral regurgitation exists—it exists in both structural damage to the valve and in dilatation due to anæmia—but whether the regurgitation depends upon causes which are in their nature permanent or removable.

We may consider this difficult question in the light of—

(*a*) History.

(*b*) Symptoms.

(*c*) Physical signs.

(*a*) A history of rheumatism is most important. No doubt, an anæmic girl may be rheumatic, and a mitral murmur present in such a case may depend on the anæmia rather than upon the rheumatism. Nevertheless, experience shows that when a mitral systolic murmur arises in association with rheumatism, it is in the great majority of cases permanent.

A history of chorea is important. A mitral systolic murmur arising in connection with chorea usually indicates permanent damage to the mitral valve, but the exceptions to this rule are neither few nor unimportant.

A history of anæmia will suggest to us the possibility of the murmur in question being hæmic in character, but must not lead us to overlook signs of organic disease.

* See p. 309.

(*b*) The symptoms will often fail to differentiate the two conditions under consideration. Dyspnœa, faintishness, and dyspepsia are common in both. Œdema of the feet and ankles may occur either in anæmia or in organic mitral disease. Dropsy of the serous cavities, cyanosis, hæmoptysis, pulmonary congestion, and albuminuria would point to the murmur under consideration being due to organic valvular defect.

(*c*) I doubt if the murmur of anæmia and of structural disease can be confidently distinguished by their physical characters alone. Nevertheless, it may be asserted that a hæmic murmur in the mitral area does not usually entirely replace the first sound, is not well conducted, and is much under the influence of posture. The contrary characters are, upon the whole, more likely to be present in structural disease.

The statement that the hæmic murmur has its point of maximum intensity, not at the true apex, but a little to the left or right of that point, is not, I think, well founded.

By far the most important guide to the differentiation of the organic and the hæmic systolic murmur in the mitral area is the state of the cardiac chambers, especially of the left ventricle. Whether anæmia ever causes recognizable hypertrophy or dilatation of the left ventricle is an open question. I think it probable that anæmia and over-exertion may cause a certain degree of dilatation, but as a broad rule the anæmic heart is not perceptibly altered in dimensions, while changes in the left ventricle are practically constant—however variable in degree—in structural mitral disease.

In connection with this difficult subject the following axioms may be suggested:—

- (1) A murmur, systolic in time, loud in the pulmonary area, weak in the mitral area, not well conducted,

unaccompanied by dilatation or hypertrophy of the left ventricle, in a patient who is anæmic and who is not rheumatic, is almost certainly functional in origin.

- (2) A murmur, systolic in time, with its maximum in the mitral area, well conducted towards the axilla, accompanied by recognizable hypertrophy or dilatation of the left ventricle, is organic in origin.
- (3) A murmur, systolic in time, limited to the mitral area, not replacing the first sound, not well conducted, unaccompanied by recognizable hypertrophy or dilatation of the left ventricle, in an anæmic subject who has not had rheumatism, is probably functional in origin, but may be organic.

Foxwell gives the following account of the state of the heart in anæmic debility: *—

“The apex beat is displaced upwards and to the left. The superficial area of cardiac dulness is increased upwards, often to the second space.

“There are three systolic murmurs—

- (a) Pulmonary—focus, junction of the third left costal cartilage with the sternum, best conducted along a line leading thence up and out to the left shoulder, not well heard to right of sternum.
- (b) Tricuspid—best heard over the third and fourth left spaces, but also well to the right of the sternum, and often with marked distinctness upwards to the second right space, and even some way along the vessels of the right side of the neck.

“Both the tricuspid and pulmonary murmurs are increased on lying down, but the pulmonary much more

* A. Foxwell, M.A., M.D.: *Essays in Heart and Lung Disease*, pp. 283 *et seq.*

than tricuspid. Both murmurs may be only audible in the supine position, though plainly so then.

- (c) Mitral—best heard at the apex, soft and not well conducted, seldom entirely replaces the first sound. Should it, however, be loud and harsh, it is quite as well heard in the axilla and at the angle of the scapula as the murmur due to organic mitral disease. It is the most fugitive of the three murmurs, sometimes occurring with every third or fourth beat only, seldom persisting for many days together, but coming and going with unaccountable vagrancy.

“The pulmonary second sound is usually accentuated in anæmic patients, if they are up and about.”

F. A systolic murmur appears in the mitral area in the course of an acute febrile illness.

How are we to determine whether it is due to temporary weakness of the myocardium and papillary muscles or to permanent organic changes in the mitral valve?

This question, which is one of much practical importance, cannot always be answered positively, but in many cases a more or less probable opinion may be formed.

If the murmur appears in the early stages of acute disease, before muscular debility and anæmia have had time to develop, there will always be a considerable degree of probability that the murmur is organic in origin. If, on the contrary, the murmur makes its appearance at a late stage of acute disease, when muscular debility and anæmia are marked, there will always be a considerable degree of probability that it is “functional” in origin, and may be expected to disappear as the patient recovers.

If hæmorrhage or persistent diarrhœa have been features of the case, the probability that a murmur

appearing in the course of the disease is functional in origin is increased.

Baginsky discusses the question whether a systolic murmur appearing in the mitral area in the course of acute disease in childhood is usually functional or organic. He quotes Hochsinger in favour of the generally functional character of such murmurs, while his own opinion inclines in the opposite direction. He is of opinion that children who suffer from functional heart disorder are usually very pallid, the arterial tension very poor, the heart sounds dull in character, and the pulmonary second sound accentuated. He advises that weight should be attached to the duration of the illness, and to such symptoms as colliquative diarrhœa and hæmorrhages.* In this connection, acute rheumatism obviously holds a special position amongst acute febrile diseases. It occasionally happens that in the later stages of acute rheumatism a systolic murmur, due to muscular debility or anæmia, appears and clears up as the patient gets well. The presumption is, however, usually to the contrary. In the large majority of cases a systolic mitral murmur appearing in the course of acute rheumatism is due to endocarditis, and will be permanent.

G. A systolic murmur appears in the mitral area in the course of chorea.

Can we decide whether it is functional or organic in origin, and give any opinion as to the probability of its disappearance?

This question does not always admit of a positive answer. The close relation of chorea to rheumatism, and the probability that symptoms of cardiac disorder appearing in the course of chorea may be due to rheumatism,

* *Lehrbuch der Kinderkrankheiten*, seventh edition, p. 767.

whether there are any articular manifestations or not, is generally recognized. Nevertheless, the cases of chorea, apparently due to fright, and not obviously associated with rheumatism, are numerous. The cause of the cardiac involvements in such cases can only be conjectured. Minute hæmorrhages of the arterioles of the endocardium of the valves have been suggested. That such cases should get well is not surprising, and it is in accord with experience that in a substantial proportion of cases (a decided minority, however, in my judgment) a systolic mitral murmur appearing in the course of chorea ultimately, perhaps after a long period, gets well. The contrary event is, however, more usual, and may be regarded as certain in rheumatic cases, and in cases where any degree of dilatation or hypertrophy of the left ventricle can be made out.

On this subject, Ashby and Wright remark: "It is seldom possible to say dogmatically that a bruit heard during the course of chorea is simply hæmic, and it is necessary to have the patient under observation for a long period during convalescence before we are in a position to say if a so-called hæmic bruit is due to organic disease or not."*

H. As the result of rheumatic endocarditis, a slight degree of mitral regurgitation, with the usual physical signs, has arisen.

What are the prospects of this lesion disappearing or becoming permanent?

In the great majority of cases the lesion will be permanent. Nevertheless, there are exceptions to this rule.

The proportion of complete recoveries is small, but the number of such recoveries is important in the aggregate.

* The Diseases of Children, second edition, p. 450.

I have met with a few cases where mitral regurgitation, dependent on rheumatic endocarditis, had been known to exist, and where after the lapse of years no trace of murmur or of hypertrophy or dilatation could be detected.

It must be borne in mind that the murmur may be temporarily absent, and may only be evoked after severe exertion. The influence of posture must be remembered.

I. A systolic murmur is heard with maximum intensity between the apex and the lower left edge of the sternum. How shall we decide whether it is due to mitral regurgitation or to tricuspid regurgitation?

Broadbent says, "The murmur attending tricuspid regurgitation is systolic in time, and is usually blowing in character, having its maximum intensity about one-third of the distance between the left edge of the sternum and the vertical nipple line. It is usually audible outwards towards the apex, and sometimes at the apex itself, where it may be mistaken for a mitral murmur." *

The differentiation of the murmur of tricuspid regurgitation from that of mitral regurgitation turns mainly on the following points:—

- (a) The seat of maximum intensity—viz. at the apex in mitral regurgitation, between the apex and the lower left edge of the sternum or over the ensiform appendix in tricuspid regurgitation, or perhaps to the right of this point.
- (b) The conduction of the murmurs—viz. into the axilla in the case of mitral regurgitation, over the right ventricle in the case of tricuspid regurgitation.
- (c) The state of the first sound in the axilla—viz.

* Heart Disease, pp. 218, 219.

more or less obscured by murmur in mitral regurgitation, clear and sometimes sharp in tricuspid regurgitation.

- (d) The coexisting symptoms. In tricuspid regurgitation we shall probably find venous pulsation epigastric pulsation, enlarged liver, ascites, cyanosis. These symptoms will not be present in mitral regurgitation, except at an advanced stage.

Tricuspid regurgitant murmurs are not uncommon. They are found both in functional and organic disease. Their significance is usually to be measured by the symptoms and by the degree of dilatation of the right ventricle.

- J. A patient presents symptoms suggestive of organic cardiac disease—*e.g.* breathlessness on exertion, præcordial oppression, weak, frequent, perhaps irregular pulse, no murmurs can be heard, the heart is not perceptibly altered in its dimensions, renal, nervous and other non-cardiac disease can be excluded.

What view are we to take of such cases? Every clinician is familiar with this problem, which often gives rise to great difficulties. Stated in different language, the problem is this—history and symptoms strongly suggest the existence of cardiac disease, physical signs are ambiguous or normal. What is the probable explanation?

These cases, which are frequent, fall in my experience into three categories—

- (a) Aggravated functional cardiac disorder.
- (b) Mitral stenosis, with disappearance of the murmur.
- (c) Myocardial change, especially fibroid and fatty degeneration.

In aggravated functional disorder there may be

breathlessness, præcordial oppression, and weak, irregular pulse, but usually these symptoms are less closely related to exertion than in the case of organic disease, the patient is probably either anæmic, neurotic, or dyspeptic; there is often a morbid subjectivity present; over-indulgence in tea, tobacco, or alcohol is common; the first sound is apt to be clear and short, but not notably intensified or altered in pitch; the second sound in the mitral area is normal; tonics, aperients, or sedatives relieve the patient's symptoms.

Mitral obstruction should be carefully borne in mind in connection with the problem under discussion. In a good many such cases this lesion is present, and if the characteristic murmur be inaudible, the diagnosis may be difficult. The female sex, youth, a rheumatic history will strengthen the suspicion of mitral obstruction, if that diagnosis seems probable on other grounds. The typical rough, vibratory, "ingravescent," pre-systolic murmur may be entirely absent during rest, may reappear after exertion, or may be absent even after exertion. A peculiar sharpness and high-pitched quality of the first sound in the mitral area may give us a valuable hint as to the presence of mitral obstruction. The second sound in the pulmonary area will usually be intensified, and the pulse will be rather small, of fair tension, and either regular or irregular.

Many of these cases under consideration depend upon myocardial changes—fatty, fibroid, or of the nature of cloudy swelling. Coronary disease often plays a large part. The relation of sclerosis of the heart to inflammation on the one hand, and on the other to coronary disease, is not yet fully made out.

Myocardial change cannot always be diagnosed with certainty. Amongst the points which serve to strengthen the suspicion of its existence are the following :—

- (a) Age—usually after 45.
- (b) A history of syphilis or alcoholism.
- (c) Dyspnœa ; anginoid, or syncopal attacks.
- (d) Marked feebleness of the heart sounds and impulse.
- (e) Excessive frequency, marked slowing, or arrhythmia of the pulse. *
- (f) Presence of renal or hepatic cirrhosis.
- (g) Intractability under treatment.

Huchard dissents wholly from the view that myocardial changes are impossible, or even difficult of diagnosis. After completing a most elaborate survey of the symptomatology of arterio-sclerosis of the heart, he speaks as follows: "*Telle est, dans ses lignes générales, la symptomatologie de l'artério-sclérose du cœur, et cette description est destinée à réfuter l'opinion des auteurs anciens et modernes qui regardent comme difficile et même impossible le diagnostic de ces sortes de cardiopathies. En s'appuyant sur le contrôle des constatations nécroscopiques, on doit affirmer que ce diagnostic est aussi facile que celui des affections valvulaires. Pour les premières nous n'avons certes pas pour nous aider l'existence de souffles révélateurs d'insuffisances ou de rétrécissements valvulaires; mais depuis la découverte de Lennec on s'est trop habitué à chercher des souffles cardiaques pour conclure à l'existence d'une cardiopathie. A ce point de vue, une réaction trop lente a commencé à se produire.*" †

I can hardly subscribe to these views.

The physical signs of valvular disease are in a large proportion of cases definitive and decisive. The physical

* Fränkel denies that arrhythmia is characteristic of arterio-sclerosis of the heart. Huchard, on the contrary, affirms that in certain cases "*l'arythmie est le symptôme prédominant de la cardio-sclérose.*"

† H. Huchard: *Traité clinique des Maladies du Cœur et de l'Aorte*, vol. i. p. 353.

signs of myocardial disease are indefinite and inconclusive, highly significant when taken as a whole, and, above all, when interpreted in the light of the history and the symptoms, but not to be relied upon singly.

One can heartily endorse Huchard's views regarding the frequency of arterio-sclerosis of the heart, upon its close relation to coronary disease, and upon the contention that since Lænnec's time we have all been too much the slaves of auscultation, too completely under the tyranny of murmurs. From every point of view, but more particularly from that of prognosis, the observer will do well, while availing himself of every evidence of valvular involvement, to ask frequently the questions—What is the probable state of the myocardium? Is coronary disease present? Can I find any evidence in kidneys, liver, lungs, or nervous system to strengthen the suspicion of important changes in the heart muscle and its vascular supply?

LECTURE XX.

PROGNOSIS IN CHRONIC VALVULAR DISEASE OF THE HEART.

SUMMARY.

Difficulties of prognosis in valvular disease.

Three types of case, viz.—

- A. The relatively favourable class.
- B. The relatively unfavourable class.
- C. The doubtful class.

Indications for assigning cases to these classes.

Analysis of two hundred consecutive cases of valvular disease occurring in hospital practice, with a view of determining their prognostic indications.

Influence upon prognosis of the following factors :—

- (a) Cause of the advent of symptoms.
- (b) Nature of the pathological process in progress.
- (c) Valve affected.
- (d) Extent of the lesion.
- (e) Condition of the cardiac chambers and myocardium.
- (f) Age and sex.
- (g) Habits, occupation, and mode of life.

THE prognosis in chronic valvular disease of the heart involves peculiar difficulties, for the following reasons :—

- (a) We cannot always form a confident opinion of the extent of a lesion, although its nature may be clear.
- (b) Prognosis depends in large measure upon the condition of the cardiac muscle, and this is difficult of determination.
- (c) Intercurrent disease—influenza, diphtheria, typhoid

fever—may modify or entirely upset our prognostic forecast.

- (d) The prudence and docility of the patient count for much. A momentary forgetfulness of our warnings, a rush for a train or a tramcar, a casual indulgence of the appetite, some emotional stress, may change the complexion of a case, and nullify the most careful and apparently well-founded prognosis.

Nevertheless, in no disease is prognosis of more vital moment than in valvular disease of the heart, in none is a judicious forecast of the probabilities of the case of more practical value, in none is it more often our privilege on the one hand to give salutary cautions, and on the other to restore hope and stimulate confidence.

For the purposes of prognosis we may divide valvular cases into three great classes, viz.—

Class A.—The relatively favourable group, *i.e.* where the existing lesion is free from present danger, and where there is a fair prospect of more or less prolonged life and activity.

Class B.—The relatively unfavourable group, *i.e.* where present danger exists, and where the prospects of a good rally under treatment are not hopeful.

Class C.—The doubtful group, *i.e.* where the outlook is simply uncertain.

It will help us much in dealing with valvular disease if we can determine, with even approximate accuracy, to which of these groups any given case should be assigned.

Our greatest difficulties will arise in connection with Class C, where there is the widest margin for error, where the prognostic indications are the least definite.

The first rule which I have to suggest is a negative one,

viz. that the question of the class to which any individual case is to be assigned cannot be determined simply by reference to the existing lesion. In other words, any valvular defect may be consistent either with a relatively good, a relatively bad, or a doubtful prognosis. No doubt, other things being equal, there is an important difference between the prognosis of, *e.g.* mitral regurgitation and mitral obstruction, or between aortic regurgitation and aortic obstruction, but we are apt to exaggerate these differences and to over-rate their prognostic significance. This question will be fully considered hereafter. In this place, I content myself with remarking that the determination of the nature of the valvular lesion present in any individual case takes us only a short way in prognosis. No error is more fundamental than the assumption that a case of a relatively favourable lesion, *e.g.* mitral regurgitation, must do well, or that a case of a relatively unfavourable lesion, *e.g.* aortic regurgitation, must do ill.

Class A.

Valvular disease of the heart, of whatever special form, admits of a relatively good prognosis when the following conditions are present, viz.—

- (a) Age, over 20 and under 50. Cases of valvular disease in childhood or early adolescence do not, as a rule, admit of a favourable prognosis, although there are numerous exceptions.* After 50, the probability that a lesion is degenerative and hence necessarily progressive has to be considered.
- (b) Absence of symptoms.
- (c) Absence of recognizable dilatation of the cardiac chambers.

* See pp. 413, 414.

- (d) Absence of vascular degeneration.
- (e) A pulse of moderate frequency and of good tension and normal rhythm.
- (f) Proof that the lesion has lasted for three or more years.
- (g) A favourable occupation, not involving undue exposure or excessive fatigue or strain.
- (h) Temperate habits and a hopeful disposition.
- (i) A good family history.*

Where the above conditions are present—and such cases are by no means rare—we are justified in giving a relatively good prognosis, *i.e.* we may assure the patient that there is no present danger, provided reasonable precautions are taken, and that there is a fair probability of a more or less prolonged period of good health and regulated activity.

A few of the foregoing points call for comment.

If symptoms be present, the earliest and most characteristic being usually dyspnoea on exertion, the case falls under Class B or C, but not under Class A. It is important to determine not only whether symptoms are actually present at the moment, but whether they have been present in the past, as this point has considerable prognostic weight. The following case illustrates this point :—

The patient, a hard-working medical man of about 45 years of age, had had aortic regurgitation, without symptoms, for several years. He pursued a laborious practice without inconvenience until a certain date, when he woke up in the course of the night with a severe attack of præcordial oppression. Rest and treatment speedily relieved his symptoms, and in four or five days he felt as well as usual. After about a week's rest he persisted in

* These are substantially the rules laid down by Andrew Clark.

resuming work, contrary to advice. About a month afterwards he died suddenly, almost without warning.

If a patient with valvular disease has no symptoms, and *never has had any*, we may feel sure that the forces at work are adequate for the maintenance of circulatory equilibrium. If the patient has had an attack of cardiac symptoms, and these have entirely disappeared under treatment, the case may, indeed, pursue a quite favourable course, but it belongs to Class C rather than to Class A. There is a distinct line of differentiation to be drawn between the two cases.

The existence of recognizable dilatation, of course, puts the case out of the category of the relatively favourable group, Class A. Not so, however, does a moderate degree of hypertrophy. Some of the best cases which have come under my observation, *i.e.* cases of prolonged freedom from symptoms and the maintenance of good general health in spite of valvular defect, have had an appreciable and sometimes considerable degree of hypertrophy of the left ventricle.

The importance of vascular degeneration from the point of view of prognosis need not be more than adverted to, as it is one of the most obvious facts in this connection. A degenerative lesion is in its nature progressive, and is commonly allied with constitutional conditions or hereditary tendencies which are only to a very limited extent under our control. The distinction between aortic regurgitation depending upon rheumatic endocarditis and aortic regurgitation depending on arterio-sclerosis is a vital one. The prognosis is much better in the former case. This point will claim our attention later on.

Proof that the lesion has lasted for three or more years may be difficult, or impossible, to obtain. Many of these cases are discovered accidentally, *e.g.* in connection with

examinations for life insurance, and there may be no data for determining the probable duration of the case. Nevertheless, it is most important to ask the question, Is this lesion probably recent or of long standing? If the former, the prognosis must always be guarded, as we have no measure of the capacity of the heart to withstand successfully the strain thrown upon it. If, on the contrary, the lesion is probably of long standing, and compensation has been successfully maintained for a considerable period, the outlook is favourable. In seeking a clue for the probable duration of a valvular lesion, we may obtain assistance from a history of rheumatism, chorea, scarlet fever, or strain, but it is evident that the information thus derived can never amount to more than a certain limited degree of probability.

The importance of occupation and habits is obvious, and need not be dwelt upon in detail. The worst occupations for the subjects of valvular disease are those which involve the double elements of strain and exposure. How far a purely sedentary occupation, by promoting muscular debility or fatty degeneration, may operate unfavourably, it is difficult to say, but the point should not be entirely overlooked. An occupation involving much exposure may render the patient liable to further rheumatic attacks, and so increase his tendency to renewed valvular implications.

From the point of view of habits, alcoholism is the most important factor. Luxurious living, in general, and the excessive use of tobacco are also unfavourable.

Family history is important. There are families where many individuals enjoy excellent health until the age of 50 or 55, and then die off suddenly from various causes, *e.g.* apoplexy, aneurism, valvular disease, renal disease. The underlying fact in such cases is premature

vascular degeneration. As Osler truly and humorously puts it, "Bad material was used in the tubing." Valvular disease arising in such families is of evil augury. The usual resisting and recuperative capacity of vessel and muscle cannot be relied upon.

With regard to the pulse, two leading facts require to be borne in mind. A very low tension pulse may indicate a weak left ventricle, while a high tension pulse involves increased arterial resistance, and increases the labour of the ventricle, already handicapped by the existing valvular defect. Cases of valvular disease do best in patients whose arterial tension is habitually somewhat low.*

Can we always assure a patient who has valvular disease without symptoms that there is no present danger?

To this question an affirmative answer may, I think, be given. I believe that in no form of valvular disease does the end ever come without warning, though the warning may be overlooked.

Class B.

We must now attempt to define the type of valvular disease in which the prognosis is relatively unfavourable, *i.e.* where present danger exists and where the prospects of a good rally under treatment are not hopeful. Once more, this question is not to be decided simply by reference to the nature of the existing lesion. The prognosis may be relatively unfavourable in any form of valvular disease.

The points of most weight in justifying a relatively bad prognosis are the following :—

(a) The nature of the symptoms. Of grave import are the following symptoms: viz. marked dyspnoea independent of exertion, cyanosis, general dropsy, anginoid and syncopal attacks, congestion of the lungs, weak, frequent,

* W. H. Broadbent: Heart Disease, third edition, p. 184.

and irregular pulse, persistent insomnia, Cheyne-Stokes respiration. Hæmoptysis and albuminuria are, upon the whole, less serious.

But these symptoms may all yield to treatment. How are we to determine that in a given case they will probably end fatally within a limited time?

This question may not always admit of a definite answer, but weight may be given to the following considerations:—

Have the above symptoms, or any considerable number of them, developed for the first time, or is this a second, third, or fourth attack? It might have been conjectured that a previous recovery from such symptoms warranted the expectation of recovery in subsequent attacks. This would not be a safe rule. A first attack of urgent cardiac symptoms is more likely to be recovered from than a second, a second than a third, and so forth. No doubt the explanation is that the reserve power of the heart gets worn out—that reserve power being in part the capacity of the heart to resist increased strain, in part its capacity of response to remedies, especially to digitalis.

Secondly, we may ask, How far has treatment had a fair trial? In no disease does the factor of “response to treatment” count for more with reference to prognosis than in valvular disease of the heart. Two cases presenting almost identical signs and symptoms may involve a totally different prognosis, because in one rest, regulated diet, digitalis, and other remedies have had a fair trial without result, while in the other the patient has been pursuing his usual avocations and has had no treatment at all. I have found this rule of the utmost practical importance.

A nice point to decide is the time limit in such cases. When can we say that treatment has had a fair trial? How soon is it justifiable to conclude that there is not

going to be any "response to treatment"? Perhaps it would be rash to attempt to answer these questions. I can only venture on the merest approximation to an answer. Usually, in these cases we expect some definite improvement, if a rally is going to take place at all, in a limited number of weeks—three, four, or five, as the case may be. The following recent case has been somewhat isolated in my experience. The patient, a woman of about 35 years of age, suffering from mitral and tricuspid regurgitation, with extreme dyspnœa, general dropsy, enlargement of the liver, and insomnia, made practically no progress under treatment for six weeks. My hopes of a rally had become very faint, when improvement slowly set in, and gradually continued until all the more active symptoms had disappeared. The expression, "a fair trial of treatment," must, then, be interpreted cautiously. Yet we shall not usually be wrong in concluding that where a good result is to be obtained in these cases improvement will not often be delayed beyond four or five weeks.

Thirdly, What is the amount of dilatation present and what is the state of the pulse?

Any extreme degree of dilatation of the left ventricle, still more of both ventricles, warrants a more or less definitely bad prognosis. Such dilatation shows that the resisting power of the myocardium is poor, or that the lesion is more than usually severe.

The pulse may give important indications. An extremely frequent, small, "thready," irregular pulse, in association with cyanosis, dyspnœa, and dropsy, generally portends a fatal issue. Intermittence is of grave augury in aortic regurgitation. Irregularity may point to a failing left ventricle, or only to disordered innervation of the heart.

The pulse is, upon the whole, a more trustworthy

guide in mitral than in aortic disease. I can hardly recall a case of mitral disease, tending to a fatal issue, where the pulse remained of fair quality. I could not make this statement with regard to aortic disease.

In aortic regurgitation I have learnt to attach much weight as regards prognosis to a sudden seizure of acute præcordial oppression, whether amounting to definite angina or not. The outlook in such cases is usually precarious. It is probable that this symptom is the index to important changes, either in the myocardium or in the coronary vessels, or both—changes which, in the present state of our knowledge, we cannot recognize by physical signs.

I should, therefore, attempt to define the type of valvular disease of the heart where the prognosis is more or less definitely unfavourable somewhat as follows: Any case where in association with valvular defect there is marked dyspnoea independent of exertion, cyanosis, increasing dropsy, and pulmonary congestion, "thready" pulse, anginoid pain, persistent insomnia, especially if much dilatation of one or both ventricles be present, and if treatment has been unavailingly tried. Some of these cases rally, and even the most desperate cases are not to be abandoned in despair, but prognosis must take account mainly of averages, and in the main the above type of case does ill.

When face to face with a case of valvular disease where danger is evidently present, it is always well to ask ourselves, Is the danger imminent or not? May this patient expire at any moment, or is it simply that the end is approaching, but may still be comparatively distant? In no disease is this distinction more difficult than in valvular disease of the heart. It is comparatively easy to say that danger, perhaps great danger, is present, but it is

often beyond the resources of our art to decide whether the end is near or not. In severe cases it is well to recognize that a sudden termination is always possible, and is by no means confined to cases of aortic regurgitation.

Class C.

To this class belong those cases of valvular disease where the prognosis is doubtful, *i.e.* where present danger cannot be excluded, but is probably not immediately urgent, where the patient may or may not respond to treatment and be restored for a longer or a shorter period to a condition of comparative health and vigour.

This is a very large class. It includes cases of every variety of valvular defect, cases which deviate only slightly from those included under Class A, or which approximate to those included under Class B. Practically it includes every case where cardiac symptoms are present but where definitely unfavourable features are absent.

Prognosis in this doubtful class turns mainly upon the following points :—

- (a) The cause of the advent of symptoms.
- (b) The nature of the pathological process in progress.
- (c) The valve affected.
- (d) The extent of the valvular lesion.
- (e) The condition of the myocardium.
- (f) Certain general considerations, *viz.* age, sex, occupation, habits, environment, family history.
- (g) Response to treatment.

(a) *The cause of the advent of symptoms.*

If we can assure ourselves that the signs of incipient failure of compensation are due to sudden strain, excessive labour, worry, emotional stress, or dietetic errors, the outlook is more hopeful than when no exciting cause can be discovered, because the above causes are removable, and

with their removal improvement of the cardiac condition may be fairly expected. The most unfavourable cases are those where dyspnoea and other symptoms insidiously arise while the patient is pursuing a well-regulated life of comparative ease and quiet and of strict temperance. In such cases we have too much reason to fear a progressive valvular lesion or degenerative changes in the myocardium.

(b) *The nature of the pathological process in progress.*

If we have reason to suspect that the primary condition is degenerative, the advent of symptoms is more ominous than where rheumatic endocarditis is the fundamental fact. The former condition is evidently more progressive; it is more apt to be associated with grave changes in the aorta, the coronary vessels, and the myocardium; the influence of age comes into play; the recuperative power of the organism is less; the probability of renal or other important visceral changes is greater.

The possibility of syphilis should be borne in mind in cases where there is evidence of vascular degeneration.

The influence of this factor upon prognosis is not, however, usually marked.

(c) *The valve affected.*

It has already been affirmed that the determination of the valvular lesion takes us only a short way in prognosis. It is fallacious to classify the various valvular lesions in their order of relative gravity, as if that order could be determined simply with reference to the lesion, and without regard to history and symptoms. A relatively favourable lesion, *e.g.* mitral regurgitation, may be accompanied by an early failure of compensation. A relatively unfavourable lesion, *e.g.* aortic regurgitation, may be attended by complete compensation and last without symptoms for many years.

That the mere determination of the valvular lesion takes us only a short way towards a just prognosis will be evident if we compare the discrepant views on this subject which have been expressed by some of the leading authorities. I will place some of these views side by side as a salutary reminder that prognosis in valvular disease is not to be decided off-hand by reference to any single factor.

Walshe writes as follows: "As a matter of clinical experience, the chief valvular derangements may be placed in the following descending series on the basis of their relative gravity, *i.e.* estimating their gravity not only by their ultimate lethal tendency, but by the amount of complicated miseries they inflict:—

Tricuspid regurgitation,

Mitral regurgitation.

Mitral constriction.

Aortic regurgitation.

Pulmonary constriction.

Aortic constriction." *

Broadbent writes: "My own experience would lead me to modify Walshe's arrangement somewhat, and to give this order of relative danger, viz.—

Aortic incompetence.

Mitral stenosis.

Aortic stenosis.

Mitral incompetence." †

Strümpell writes: "In regard to the particular forms of valvular disease, aortic insufficiency generally gives the best prognosis, inasmuch as it may be very perfectly compensated for many years, but if severe disturbance of compensation once occurs in this form of heart disease, it gives a very unfavourable prognosis, since as a rule we

* Diseases of the Heart, fourth edition, p. 393.

† Heart Disease, third edition, p. 79.

cannot reinvigorate the heart. Mitral insufficiency is, also, quite a favourable form of heart disease, which may be compensated for a long time. Mitral stenosis is decidedly more unfavourable in its prognosis. . . . Aortic stenosis is also capable of quite good compensation, and in this respect it is even more favourable for the patient than mitral stenosis." *

Osler writes: "Aortic insufficiency is unquestionably the most serious (*i.e.* of forms of valvular defect). . . . Aortic stenosis is a comparatively rare lesion . . . and is, as a rule, well compensated. In mitral lesions the outlook, on the whole, is much more favourable than in aortic insufficiency. Mitral insufficiency, when well compensated, carries with it a better prognosis than mitral stenosis." †

Allbutt agrees in the main with Broadbent. He says: "The course of aortic regurgitation is towards death. Let sanguine prophets say what they may, ten years is a long time in any case of aortic insufficiency. . . . Mitral insufficiency is the only heart disease which, under favourable circumstances, can be nursed to an indefinite duration." ‡

Wilks § thinks that mitral stenosis is a more favourable lesion than mitral regurgitation, an opinion not generally entertained at the present day.

The student will probably be somewhat discouraged by this conflict of opinion. From it he may, however, learn a useful lesson, *viz.* that prognosis in valvular disease is a difficult matter, and depends upon many other considerations as well as upon the nature of the lesion.

With the view of obtaining some information regarding the relative gravity of the different valvular lesions, I have

* A Text-book of Medicine, English edition, p. 334.

† Practice of Medicine, fourth edition, p. 729.

‡ System of Medicine, vol. v. pp. 953, 954, 955.

§ Hilton Fagge's Principles and Practice of Medicine, second edition, p. 967.

prepared the following analysis of 200 consecutive cases of heart disease occurring in my practice at the Royal Victoria Hospital, Belfast.

Age.		Mitral obstruction.	Mitral regurgitation.	Aortic regurgitation.
10-20	...	5	16	6
20-30	...	13	31	5
30-40	...	5	30	14
40-50	...	1	18	9
50-60	...	0	18	6
60-70	...	0	16	6
70-	...	0	1	0
		24	130	46

FATAL CASES (23).

10-20	...	1	1	0
20-30	...	1	1	0
30-40	...	0	6	1
40-50	...	0	2	1
50-60	...	0	3	2
60-70	...	0	3	1
		2	16	5

How many had passed the age of 40?

Mitral obstruction, 1 in 24.

Mitral regurgitation, 53 in 130, *i.e.* 1 in 2'4.

Aortic regurgitation, 21 in 46, *i.e.* 1 in 2'2.

How many had passed the age of 50?

Mitral obstruction, none.

Mitral regurgitation, 35 in 130, *i.e.* 1 in 3'7.

Aortic regurgitation 12 in 46, *i.e.* 1 in 3'8.

How many had passed the age of 60?

Mitral obstruction, none.

Mitral regurgitation, 17 in 130, *i.e.* 1 in 7'6.

Aortic regurgitation, 6 in 46, *i.e.* 1 in 7'6.

How many had passed the age of 70?

Mitral obstruction, none.

Mitral regurgitation, 1 in 130.

Aortic regurgitation, none.

It must be observed that the above figures represent *minimal* averages. They are based on the facts obtained in patients still living. Many of these, no doubt, survived for considerable periods after their treatment in hospital. Average age during life is one thing. Average age at the time of death is, of course, quite a different matter. I have inquired into this latter point, but the numbers are hardly sufficient to warrant any secure conclusions. Two deaths in mitral stenosis do not afford any basis for deduction. The average age at time of death in 16 fatal cases of mitral regurgitation was 42 years—in 5 fatal cases of aortic regurgitation, 45 years.

I have not included aortic obstruction in the above catalogue, as my experience of this rare lesion is quite insignificant.

The number of deaths in each lesion is no secure measure of its gravity, as in hospital practice it is often a matter of mere accident whether cases of heart disease visibly approaching the close will return home to die or await the end in hospital.

More light may be obtained by investigating the relation of age to the various lesions.

It is evident that few cases of mitral obstruction pass the age of 40. I have seen two cases of this lesion at the age of 49 and one case over 50. Such cases are, however, exceptional. According to Broadbent, the average age at time of death in this lesion is 33 in the case of males, and 37 or 38 in the case of females.* My returns would tend to corroborate this computation.

* Heart Disease, third edition, p. 211.

In marked contrast is the case of mitral regurgitation, of which no less than 53 cases out of 130, or one in 2·4, passed the age of 40. The conclusion that the latter is the more benign lesion can hardly be resisted. The comparison between these two lesions as regards the age limit is a fair one, inasmuch as there is no reason to suppose that mitral obstruction arises, on the average, earlier or later in life than mitral regurgitation.

The comparison between mitral regurgitation and aortic regurgitation is, from this point of view, less secure, inasmuch as the former lesion arises most often in early life, while the latter lesion is frequently degenerative and often begins at or after middle life. Thus, when we find that 35 cases out of 130 of mitral regurgitation passed the age of 50, and 17 out of 130 passed the age of 60, and that the proportion of cases of aortic regurgitation passing these age limits is about the same (viz. one in 3·7 and 7·6 in the case of mitral regurgitation, and one in 3·8 and 7·6 in the case of aortic regurgitation), it does not follow that the duration of these two lesions is approximately equal. A much larger proportion of the mitral cases than of the aortic have undoubtedly dated from the earlier years of life.

In advanced life, mitral regurgitation practically holds the field. It is not very rare to find it in patients at or over 70.

We may venture on the following summary of the prognostic indications in the chief valvular lesions.

Mitral obstruction.

Upon the whole, a severe lesion, although often well compensated for many years; will probably terminate life in or before the fourth decade; prognosis depends largely on the staying power of the right ventricle; cyanosis, dropsy, and disappearance of the second sound at the

apex are of evil augury ; arrhythmia of the heart is not necessarily unfavourable ; the possibility of embolism, pulmonary infarction, and hæmoptysis must be borne in mind.

The rallying power of the heart after decided failure of compensation is less in this lesion than in mitral regurgitation, but greater than in aortic regurgitation.

Mitral regurgitation.

Upon the whole, a relatively favourable lesion, but of very variable gravity ; is consistent with indefinite duration, and may last into old age ; sometimes, but only in rare cases, entirely disappears ; prognosis is in definite relation to the amount of dilatation present ; a moderate grade of hypertrophy is not unfavourable ; the pulse gives important prognostic indications ; arrhythmia is more significant than in the case of mitral obstruction ; a first failure of compensation is usually followed by a good rally under treatment ; dropsy is less unfavourable than in the case of mitral obstruction.

Aortic regurgitation.

Upon the whole, a relatively unfavourable lesion, but many cases of rheumatic causation in young subjects enjoy perfect compensation for a considerable number of years ; a duration of ten to fifteen years is not very rare ; cases of degenerative origin tend to progress steadily, but a considerable number pass the age of 60 ; after a decided failure of compensation, the rallying power of the heart is deficient, and a fatal issue is always possible ; sudden death never occurs without some, though perhaps slight, warning ; unfavourable features are a markedly collapsing quality of the pulse, disappearance of the aortic second sound in the neck, anginoid and syncopal attacks,

marked vertigo, persistent insomnia, decided anæmia, Cheyne-Stokes respiration.

(d) *The extent of the lesion.*

The determination of the extent of damage to a valve is a matter of considerable difficulty. The following considerations may assist in forming an estimate of the probable amount of reflux or constriction.

In aortic regurgitation a large amount of reflux is indicated by a decidedly collapsing quality of the arterial pulse, the disappearance of the second sound in the neck, marked dilatation of the left ventricle, and symptoms of cerebral anæmia. In the absence of these symptoms, we may conclude that the amount of reflux is moderate or small.

In aortic obstruction, a large amount of constriction is indicated by a prolonged heaving impulse, a systolic thrill, considerable hypertrophy of the left ventricle, and a small, infrequent pulse.

In mitral regurgitation, a large amount of reflux is indicated by a murmur replacing the first sound, and well conducted into the left axilla and round to the left back, by decided dilatation of the left ventricle, by changes in the right heart, by signs of systemic engorgement, and by a small, frequent, and irregular pulse.

In mitral stenosis, a large amount of constriction is shown by marked changes in the right heart, by feebleness or disappearance of the second sound at the apex, cyanosis, pulmonary congestion, signs of systemic engorgement, and a small, frequent, and irregular pulse.

(e) *The condition of the cardiac chambers and myocardium.*

As already stated, a moderate degree of hypertrophy

of the cardiac walls is compatible with a favourable prognosis, while dilatation is always unfavourable.

Dilatation of the left ventricle is shown by extension of dulness to the left, a weak, fluttering impulse, the first sound at the apex may be short and sharp, or obscured by murmur, gallop-rhythm is frequent, the pulse is small, frequent, irregular, and unequal.

Dilatation of the right ventricle is shown by extension of dulness to the right or left, impulse weak or absent, sometimes a systolic murmur in the tricuspid area, and usually visible pulsation in the veins.

Dilatation of the auricles cannot be made out with certainty.

Hypertrophy of the left ventricle is shown by a strong heaving impulse.

Hypertrophy of the right ventricle is shown by an impulse transmitted to the sternum and by epigastric pulsation.

Hypertrophy of the auricles cannot be made out with certainty.

Changes in the myocardium are of vital moment as regards prognosis. Their diagnosis is often a matter of much difficulty. The symptoms of myocardial degeneration cannot always be distinguished from those of dilatation.

The following group of symptoms is suggestive of myocardial disease:—Feeble, slow, or very frequent or irregular pulse, dyspnœa on exertion, anginoid, syncopal, epileptiform or apoplectiform attacks, Cheyne-Stokes respiration. Age and history may assist the diagnosis.

I shall conclude our survey of the subject of prognosis in valvular disease by dwelling on a few general considerations.

Age.—Cases arising in childhood, are, on the whole,

unfavourable.* The causes of this somewhat unfavourable outlook in the young are manifold. In spite of the fact that hypertrophy takes place readily at this age, the case rarely does very well, inasmuch as the disturbance of the cardiac function interferes with normal growth and nutrition, the child is less tractable than the adult, suffers more from confinement and inaction, is more prone to recurrences of rheumatism involving fresh valvular implication. Cases arising in adolescence and early adult life are, perhaps, the most favourable, inasmuch as nutrition is probably at its maximum; compensatory hypertrophy may be expected, and degenerative change is improbable.

In senile heart disease the prognosis is generally unfavourable. Degenerative changes are probably at work, compensation is inadequate, visceral complications are common, myocardial implication is probable, the response to treatment is imperfect. Nevertheless, there is a certain advantage in the diminished strain which is the melancholy privilege of senility, and in the absence of angina pectoris, renal disease, advanced arterial degeneration or unequivocal signs of myocardial changes, the prognosis should not be definitely bad.†

Sex.—Women bear valvular disease, upon the whole, better than men. This is not to be explained by any inherent sexual difference, but as related to habits and mode of life. The influence of pregnancy and parturition is unfavourable, but it is, perhaps, less potent than has been often thought.

Habits and mode of life.—This is a factor of the first importance, but the subject need not be laboured at length, as most of the points in this connection are more or less

* Henoch takes the opposite view. The point is deserving of further study.

† For some good remarks on prognosis in senile heart conditions see Balfour : *The Senile Heart*, pp. 287 *et seq.*

obvious, and have been already considered. Exposure, privation, strain, laborious occupations, alcoholism, emotional stress, liability to infection, are among the most unfavourable points.

The military life is unfavourable, so is the medical career. The clerical and the legal careers are relatively favourable. The worst occupations are those which combine the elements of exposure, strain, and hardship.

In summarizing our experience of the course of cases of valvular disease, we have to acknowledge that the unforeseen and the *unforeseeable* play a large part. Our rules must be interpreted with many grains of salt. At best, we must expect to be often at fault. But, on the whole, the more hopeful view of these cases now prevalent is fully justified. How far this more favourable view is due to prompter diagnosis, greater care in the management of conditions (*e.g.* endocarditis) which cause valvular changes, or to the general use of that potent and admirable drug, digitalis, would be interesting subjects for speculation, but their discussion would at present take us too far.

LECTURE XXI.

THE TREATMENT OF VALVULAR DISEASE OF THE HEART.

SUMMARY.

A. Treatment of Functional Disorders.

Dietetic measures, change of air, hydropathy, strychnine, arsenic, bromides, valerianates, quinine, aconite, psychical influences ; management of insomnia.

B. Treatment of Valvular Affections during the Stage of Complete Compensation.

Regulation of habits, and amusements ; question of occupation, and of marriage.

C. Treatment of Valvular Affections during the Stage of Failing Compensation.

Rest and exercise ; diet ; drugs—digitalis, indications for its use, dosage, combinations with the remedy, drawbacks to its use, effects of the remedy, digitalis in aortic regurgitation ; question of blood-letting ; strophanthus, caffeine, convallaria, spartein, cactein.

D. Treatment of Special Symptoms in Valvular Disease.

Dropsy, dyspnœa, insomnia, palpitation, præcordial oppression, dyspepsia, renal congestion.

Question of alcohol, diffusible stimulants, nitro-glycerine, arsenic, chloral.

Change of climate, "resistance" and other exercises, baths.

The Nauheim treatment.

Oertel's Terrain-Kur.

Drawbacks to special sanatoria in cardiac cases.

WE shall consider the subject of the treatment of valvular diseases of the heart under the following heads:—

A. The treatment of functional disorders.

B. The treatment of organic valvular disease during the stage of complete compensation.

- C. The treatment of organic valvular disease during the stage of failing compensation.
- D. The treatment of special symptoms.

A. The Treatment of Functional Disorders.

My experience of these troublesome affections is that we must seek their relief by the limitation of work and worry, the correction of injurious habits, the regulation of diet, by rest and change of air, and only in a minor degree by drugs.

The patient usually has either a nervous heredity, or has exhausted himself by work or pleasure, or is dyspeptic, or indulges himself too much in tea, coffee, tobacco, or alcohol, or has been the subject of emotional stress. The treatment must be arranged accordingly.

Means to brace the faltering nervous system and to regulate the disordered digestion will, in most cases, prove our most potent resource, and must be devised on general principles. The diet must be light and digestible, but mainly of proteids. The state of the teeth and the efficiency of mastication must not escape our attention. Moderate purgation will often be found beneficial. These patients bear mercurials and drastic purgatives badly, but will be found to tolerate moderate doses of aloin, cascara, or liquorice powder.

Tea, coffee, and tobacco should be forbidden in severe cases, and strictly limited in all.

Alcohol has its uses, but is a risky ally.

Fresh air, rest, mental distraction, and change are highly important. The best air for these cases is, in a majority of cases, upland air—*e.g.* Harrogate, Buxton, Crieff. Sea air is often injurious. Hot, moist, and relaxing resorts are to be avoided.

Hydropathy has only a limited utility, and if pursued

with great vigour is sometimes disastrous. The tepid douche is probably the best form of bath. Hot and prolonged baths are hurtful. In mild cases a little cautious sea-bathing may be allowed, but only when experience shows its suitability.

The most generally useful drug is strychnine. This valuable remedy should be given in small doses, and only in cases where there is not much nervous irritability. In proportion as the symptoms are atonic or torpid, strychnine will probably be found of service, while if excitement, irritability, or insomnia be present, this remedy should be avoided. Arsenic is, perhaps, the next remedy in order of utility. It shines most conspicuously where the neurotic element is prominent and is allied with anæmia.

The bromides are valuable, but should be used with a parsimonious hand. The tendency at present is to use these drugs too freely, and in cases where a tonic regimen is to be preferred. They are of special service where palpitation, heart hurry, nervousness, and insomnia are features. The combination of bromides with belladonna is sometimes a good one.

Digitalis and strophanthus are not to be advised in functional disorders.

Ammonia, ether, camphor, valerian, and asafoetida are sometimes useful, and should not be forgotten. The combined valerianates of iron, zinc, and quinine have sometimes seemed to me to be efficacious.

Quinine does not, as a rule, shine in functional heart disorders, but its general tonic and appetising properties may sometimes be brought usefully into play.

Allbutt advises the cautious use of aconite in certain cases where palpitation is a prominent feature.*

Psychical influences are potent in connection with these

* System of Medicine, vol. v., Functional Disorders of the Heart.

cardiac neuroses, and if hypnotic suggestion could be regularized and temperately used, there can be little doubt that it would be beneficial. Unhappily, the patient often virtually hypnotizes himself and invariably to his hurt. Sometimes the emphatic assurance that nothing really serious is the matter, the resolute recommendation of work, and the prohibition of further visits to the physician have an excellent effect. Many of these patients are to be cured, not by systematic treatment, but rather by the absence of it, by continuous occupation, mental distraction, and the avoidance of morbid introspection.

Insomnia is sometimes a feature of the worst cases, and calls for much discrimination in its management. Opium, chloral, sulphonal, and trional should be withheld as long as possible, but sometimes it is impossible entirely to dispense with their aid. Hyoscine is a valuable remedy, but must be used with much caution. If possible, we should treat the insomnia indirectly, rather than directly, *i.e.* by measures aimed at the general constitutional condition.

B. The Treatment of Valvular Disease during the Stage of Complete Compensation.

A "masterly inactivity" should be the rule in such cases. Often these lesions are detected by accident—as in examination for life assurance—and it is most important to bring the patient to a just view of his condition, to convince him that there is no present danger, and that active treatment is unnecessary and might be injurious.

The chief points to be borne in mind are the avoidance of excessive strain, severe labour, too strenuous amusements, unhealthy excitement, and imprudence in eating and drinking.

If the patient's occupation is one involving severe

muscular strain and much exposure, the question of its abandonment may have to be entertained.

Amusements must be regulated. Mountain-climbing, football, swimming, and, perhaps, cricket should be interdicted, while golf or bowls may be allowed. The question of cycling for these cases is a difficult one. Moderate cycling on fairly level roads at an easy pace is, I think, permissible, but hard riding, "scorching," and the like are certainly dangerous. Much depends upon whether we can trust our patient to ride quietly and moderately. If we have any doubt on this point, cycling should be entirely prohibited.

If the patient is gouty or rheumatic, the treatment appropriate to these conditions should be enforced.

The diet should be simple. Alcohol and tobacco should be reduced to a minimum or altogether prohibited.

Drugs should be sparingly used. Strychnine, iron, arsenic, and bitter tonics may sometimes be employed with advantage. Digitalis and strophanthus should be avoided.

If bronchitis or rheumatism be present, a warm, dry climate may be advised with advantage.

The question of choice of occupation and of marriage will often arise in connection with these cases. A patient with organic valvular disease should not select the career of a soldier, a sailor, or an engineer. Law and divinity are permissible avocations. The medical profession is, upon the whole, unsuitable, but some of the special branches may be selected with safety. The career of the scholar, author, or scientific investigator may be approved. The political career should be avoided.

The question of marriage is often a difficult and delicate one. In my experience, it is seldom decided on medical issues alone.

Marriage need not be interdicted to any man, whatever his valvular defect, who is for the time being strong and well, whose occupation is not obviously unsuitable, and who is able to make a livelihood. The prospects of the duration of life should, if our advice is asked, be plainly stated. Candour in such cases is imperative.

In the case of women, there are, of course, special risks. I am inclined to think that marriage should be interdicted to women who are the subjects of aortic regurgitation or mitral obstruction. In well-compensated cases of mitral regurgitation without perceptible dilatation, marriage may be allowed. The risks of parturition are often successfully surmounted in such cases. Frequent child-bearing is generally, however, hurtful.

C. The Treatment of Valvular Disease attended by Failure of Compensation.

The first question to be considered is that of rest or exercise. The due apportionment of rest, exercise, and labour is amongst the most important, and also the most difficult, problems in connection with valvular disease. With commencing failure of compensation rest is, for a time, imperative. How long it shall continue, whether it shall be absolute or not, how soon it may give place to graduated exercise or mitigated labour, are questions which the physician's knowledge and common sense must answer. No hard and fast rules can be laid down. In spite of all that has been said and written in recent years regarding the value of regulated exercise in valvular disease, there can be no doubt that with commencing failure of compensation rest should have priority. Exercise which causes any degree of dyspnoea is probably hurtful. It is true that in some of these cases the patient can "walk off his shortness of breath," but it must be a matter

of serious question whether this result is due to the heart rallying itself for the labour put upon it or simply to blunting of its sensibility. On the whole, while the cardiac patient is any degree scant of breath, let him rest. We shall never regret the adoption of this broad clinical rule. We shall often regret its breach. When compensation has been restored, the question of exercise and work becomes vital.

Secondly, the dietetic treatment of these cases is very important. If dyspeptic symptoms be slight or absent, and no serious degree of gastric catarrh exist, the diet should be composed of solid elements, and not too remote from the diet of health. It should, also, be somewhat dry. Again and again I have seen cardiac cases improve rapidly when taken off their slops of beef-tea, chicken-broth, arrowroot, and the like, and put upon a limited diet of fish, chicken, tender meats, stale bread, boiled eggs, and green vegetables. It is indispensable to secure that the meals shall be moderate or small in quantity, slowly eaten, and that mastication shall be thorough. Limitation of fluids is important, as a sloppy, watery diet increases the amount of fluid in the veins and aggravates the toils of the labouring ventricles. On the other hand, the restriction of fluids may tend to the accumulation of the products of nitrogenous waste, and must be regulated with discretion. If the urine be scanty, very acid, and of high specific gravity, the temporary use of diluents may be necessary. Still, as a broad rule, let the diet of the average cardiac case be fairly dry. If, however, gastric catarrh be a prominent feature, and discomfort after food, pain in the epigastrium or vomiting be present, it is evident that the above rules will call for modification. A light diet of milk, simple puddings, beef juice and the like will be required for a time, but it must always be

remembered that a carbo-hydrate diet tends to gaseous distension of the stomach and acidity, and that these factors do much to aggravate the sufferings of cardiac patients. If gout be present, nitrogenous foods must, of course, be limited.

As regards drugs in failing compensation, *digitalis* is our sheet anchor. This admirable remedy—one of the most remarkable in the pharmacopœia—fulfils three conditions. It increases the force of systole, prolongs diastole, and raises the tension in the arterioles. It is indicated in every case of failing compensation, with a few reserves to be mentioned presently, and we shall usually be more profitably employed in endeavouring to understand *digitalis* and frame rules for its use than in trying to find substitutes for it. All other cardiac tonics, *strophanthus* included, possess only a very subordinate value. How often am I told that *digitalis* has been tried and has failed, or that it cannot be tolerated when some, perhaps slight, variation in the form of the drug or the combinations with it serves to mitigate its drawbacks and develop its virtues. The gastric disturbance so often complained of in connection with the administration of *digitalis* is sometimes attributable in a measure to the drugs, *e.g.* carbonate of ammonia, iron, squill, mercury, usually given in association with it. Only those who make a practice of habitually employing *digitalis* by itself can thoroughly appreciate its value. In presence of the usual clinical picture of commencing failure of compensation, we shall do well to remember that for this condition we possess one sovereign remedy, and only one, *viz.* *digitalis*, and that the drugs commonly combined with it possess only a limited utility.

As regards the form of the drug, the tincture is the usual favourite in the British Islands and in America,

while the powdered leaf finds more favour on the Continent of Europe. I incline to the latter preparation, and usually prescribe it in pills of 1 gr. or $1\frac{1}{2}$ gr. combined with a little extract of gentian. These pills, or the same amount of the leaf in the form of powder, may be given twice, thrice, or four times daily according to circumstances. Strümpell advises a much more vigorous administration of digitalis. "Much experience," he says, "has shown that it is best to give it at regular intervals of two hours, in doses of one grain and a half of the powdered leaf, either in capsules or simply in water; so that the patient receives in the course of a day some five or six powders, *i.e.* eight or ten grains of digitalis. If the remedy is well borne we may continue it in this manner, but usually it is omitted during the night, to be resumed in the same way on the next day. As a rule, the distinct specific influence of digitalis is evident after the employment of ten to fifteen powders in the course of two or three days."*

Under this treatment the ventricles in many cases rally, the heart's action becomes slower, stronger, and more rhythmical, the arterial tension rises, the excretion of urine is increased, the dyspnœa is relieved, and the general clinical condition improved. The views of Douglas Powell are somewhat different from those of Strümpell. According to him, "the most common mistake which one observes in the use of digitalis is that too large a dose is prescribed at first, which tends to premature arterial contraction and cumulative effects. Then with the appearance of physiological symptoms the drug is stopped and some other medicine substituted until the pulse again calls for its administration. In this haphazard way of using digitalis the heart is never held in good control. It is alternately checked and allowed to run away again. In

* A Text-book of Medicine, English edition, p. 336.

exceptional cases, where there is urgent need to push the drug, digitaline is best used subcutaneously. In other cases a dose of 10 m. of the tincture every four hours, or 15 m every eight hours, or 5 m every waking hour, is sufficient." *

The most brilliant results from the use of digitalis which have come under my observation have been from 1½-grain doses of the powdered leaf two, three, or four times daily. I have seldom seen the drug administered in this way disagree, and I have absolutely no experience of decided cumulative effects.

We gauge the effects of digitalis mainly by watching the pulse and the urine. If the pulse improves and the urine becomes more copious, we are assured that the remedy is doing good. If, on the contrary, the arrhythmia of the heart is increased by the use of digitalis and the excretion of urine is diminished, it is evident that the drug is unsuitable. The chief drawback to digitalis is its tendency to cause gastric derangement. The advent of signs of gastric irritation suggests, not that the drug should be forthwith abandoned, but rather that the dose should be diminished, or the intervals between the doses lengthened, or that a mercurial purge should be given. If the gastric symptoms persist, the drug should be suspended for a time and then tried again. Of the various combinations with digitalis, the most important are mercury and iron. There is no doubt that blue pill or calomel is often a most important adjuvant to the action of digitalis. The well-known Guy's or Bailey's pill is a valuable combination, but I am inclined to doubt whether the squill which it contains is essential to its efficacy. On the whole, however, the habitual association of digitalis and mercury is not a convenient mode of administration. These drugs are better

* Treatment in Diseases and Disorders of the Heart, pp. 72, 73.

given separately. A few grains of blue pill or calomel, followed by a saline, every three or four days during a course of digitalis will be found to yield better results than the regular use of Guy's pill. The combination of iron with digitalis is a frequent and valuable one.

Strychnine and nux vomica are useful remedies in valvular disease, but not, I think, when given in the usual tonic doses. They are chiefly of value in emergencies, and should then be given hypodermically. Of the value of strychnine administered in this way and in fairly full doses there cannot be any doubt. Balfour recommends that in dealing with the senile heart digitalis should be combined with iodide of potash with the view of "unlocking the arterioles and promoting the free passage of blood to the veins." *

The special indications for the use of digitalis are, according to Broadbent, "frequency, weakness and irregularity of pulse, and œdema of the extremities, with scanty, turbid, and concentrated urine."† In the absence of these symptoms the efficacy of digitalis is much less conspicuous. It is not often of signal service when dropsy is absent, and if the pulse be of moderate frequency and of good tension the remedy is not indicated. Where arrhythmia is a prominent feature and dropsy is absent, digitalis is not usually of decided service.

As regards the valvular indications for the use of this remedy, digitalis achieves its most signal triumphs in cases of mitral regurgitation, and in cases of aortic regurgitation with secondary mitral reflux. It is much less useful in aortic obstruction and in mitral obstruction. In the latter lesion there may be circumstances in which the increased force of the systole of the right ventricle induced by digitalis

* The Senile Heart, p. 266.

† Heart Disease, third edition, p. 123.

may be distinctly hurtful. The case of aortic regurgitation requires fuller consideration, as there is a widespread prejudice that the use of digitalis in connection with this lesion is of doubtful propriety. I take it that this prejudice is, upon the whole, without adequate foundation. The objections which have been raised to the employment of digitalis in aortic reflux are, in the main, theoretical. It is argued that to prolong the diastolic period, during which the regurgitant flow of blood is entering the left ventricle must be to the patient's disadvantage. But how are we to decide whether the increased force of systole, also due to digitalis, may not more than counterbalance this drawback? Only clinical experience can settle the question. So long as the symptoms of aortic regurgitation are definitely "aortic" in type—viz. præcordial oppression, anginoid pain, syncopal seizures, giddiness, anæmia—digitalis is of doubtful value; but when to the above symptom-complex are added other symptoms which may, without abuse of language, be called "mitral"—viz. dyspnœa, œdema, scanty urine—then digitalis is often of signal value, and that, too, whether the auscultatory signs enable us to affirm definite secondary mitral reflux or not.

In mitral obstruction digitalis is often disappointing. So long as the right heart seems fairly vigorous, and there is neither pulmonary congestion nor dropsy, digitalis will not be found of much real service. As already mentioned, there may be cases of mitral obstruction where increased driving power of the right ventricle will only aggravate the labours of the struggling left auricle.

The drawbacks to digitalis may be divided into two classes, viz.—

- (a) Those which appear early and which are due either to over-dosage or to intolerance of the remedy on the part of the patient; and

- (b) Those which appear late and which are due to too long persistence with the remedy.

Among the inconvenient symptoms which have been attributed to the use of digitalis may be enumerated: nausea, vomiting, diarrhœa, pallor, faintness, slow and irregular pulse, diminution in the secretion of urine, hæmaturia, præcordial oppression. Huchard thinks digitalis may provoke an attack of cerebral hæmorrhage or cerebral embolism.* Some authorities advise that we should always begin with minute doses and feel our way with the remedy.

I cannot help thinking that these fears and cautions are somewhat excessive. If digitalis be given in moderate doses, the diet regulated, the state of the bowels attended to, and rest enforced, I think the cases of intolerance will be few. As to the bugbear of "cumulative effects," my experience entirely endorses the statement of Osler: "there is no such thing as a cumulative action of the drug manifested by sudden symptoms."† I am in the habit of advising a temporary suspension of the drug after a course of four to six weeks; hence, I have no experience of the ill effects of a too prolonged administration of the remedy. While the precaution of occasionally interrupting the use of digitalis is a good one, many patients can take the drug uninterruptedly for prolonged periods. In such cases the dose need not exceed one or two grains per diem. Whether the intensive mode of administering digitalis, *i.e.* by bringing the patient rapidly under the full influence of the drug, is to be approved is a moot point. German and Austrian physicians advise this course, while British experience is, upon the whole, against it. That this method of treatment may sometimes be strikingly successful I

* *Traité Clinique des Maladies du Cœur et de l'Aorte*, vol. i. p. 492.

† *Practice of Medicine*, fourth edition, p. 723.

make no doubt, but it increases the danger of causing some of the inconvenient and undesirable effects of the remedy.

In some cases the abstraction of a few ounces of blood, either by venesection or leeching, may be necessary before digitalis can exert its full therapeutic effect. The special indications for blood-letting are extreme dyspnœa and orthopnœa, with cyanosis and distension of the right heart.

Of special cardiac remedies, strophanthus comes next to digitalis, but *longo intervallo*. My experience of this remedy has been, upon the whole, disappointing, but it is necessary to add that I have seldom employed it except in cases where digitalis had been tried and had failed. This is, perhaps, hardly to give the remedy a fair chance, but the result of my limited use of strophanthus in this manner has been to convince me that where digitalis fails strophanthus seldom succeeds. In mitral obstruction, where digitalis so often disappoints us, strophanthus is sometimes of benefit. The combination of the two remedies has sometimes seemed to me very advantageous.

Caffeine is a drug which may find a place in the therapeutics of valvular disease, but I have seldom seen any brilliant results from its use alone.

Convallaria, spartein, cactein, casca, are drugs of which I have a very limited experience, and that experience has not been such as to encourage me to make any large use of these remedies. Once more I have to reiterate my conviction that, in the treatment of failing compensation dependent on valvular disease, our main reliance, so far as drugs are concerned, must be upon the judicious use of digitalis.

D. The Treatment of Special Symptoms.

This must always be subordinate to the treatment of the general condition.

Dropsy must be combated chiefly by the use of the cardiac tonics and by purgation. Saline purgatives are well borne in these cases, and may be given even when prostration is marked. Diuretin is sometimes useful. Diaphoretics are of little avail, and hot baths and hot-air baths are not suitable for cardiac cases. Puncture of the lower limbs may be practised when dropsy is marked and the skin tense. It is best performed by making a number of punctures with a darning-needle, and covering the part with flannel bandages rung out of hot water and lightly applied. With strict attention to cleanliness this is a perfectly safe procedure, and the amount of fluid thus removed is often remarkable. Southey's tubes may be used, but do not present any decided advantage over the foregoing simple method. If hydrothorax is present to any serious extent, tapping should be performed. The advantage of this procedure in cardiac cases is not always sufficiently appreciated.

Dyspnœa must be treated on lines appropriate to the general condition. If any marked increase of dyspnœa takes place, it is well to be vigilant for pulmonary complications, *e.g.* hydrothorax, œdema of the lungs, general bronchitis. For hydrothorax, as already stated, the proper treatment is tapping. For œdema of the lungs vigorous stimulation should be employed, and locally the application of mustard plasters or of dry cupping is sometimes useful. Bronchitis should be treated on general lines, but depressing expectorants must be withheld.

Insomnia is often the source of the patient's greatest suffering, and is frequently intractable. It has some relation to the amount of dyspnœa or orthopnœa present. In

addition to the general treatment, we may seek to mitigate insomnia by postural arrangements—some patients sleep best in an easy-chair—by hot applications to the præcordium, and by alcohol and morphia. Osler recommends paraldehyde. The bromides are seldom of signal service, and sulphonal and trional must be used sparingly. The hypodermic injection of morphia is by far the most effectual remedy, and there is ample evidence that it is safe. Dr. Little, of Dublin, recommends the following solution for use in these cases :—

Morphin. sulph.	gr. iv.
Chloral hydrat.	gr. ij.
Liq. atropin. sulph.	m. xii.
Aq. camphor.	ad ʒ iv.

Ten minims of this solution contain $\frac{1}{6}$ grain of morphia and $\frac{1}{240}$ grain of atropine. Little advises eight minims for a first injection in a cardiac case in an adult, less in a child. The chloral is added merely to make the solution keep, and may be omitted if the quantity ordered is for early use.*

Cough and hæmoptysis do not call for special measures in valvular disease. The latter is often salutary, and is rarely a source of danger.

Palpitation and præcordial “anxietas” are sometimes troublesome features, especially in aortic regurgitation. If we have a throbbing and hypertrophied left ventricle in these cases, the mitral valve remaining competent, digitalis will not avail us. Aconite is sometimes given in such conditions, but I have reason to regard it as a dangerous remedy. The best drug is iodide of potash, which may be combined with mercurial purgation, ice to the chest, and the cautious use of sedatives.

* Jas. Little, M.D., *Chronic Diseases of the Heart*, p. 41.

Gastric symptoms must be controlled chiefly by the regulation of the diet.*

Renal congestion calls for saline or mercurial purgation. Diuretics may be combined with cardiac tonics. The diet should not consist too exclusively of proteids, and the use of diluents may be temporarily advisable.

The value of nitroglycerine in valvular disease is a moot point. Its value in anginoid states is, of course, indisputable, but it is often given in valvular disease simply for the relief of dyspnœa. I believe this to be very doubtful practice. Osler, however, writes as follows: "There are instances of cardiac dyspnœa, unassociated with dropsy, particularly in mitral valve disease, in which nitroglycerine is of great service, if given in the 1 per cent. solution in increasing doses. It is especially serviceable in the cases in which the pulse tension is high."† It is clear that this powerful remedy can only aggravate the patient's condition if arterial anæmia is present. Allbutt says, I think rightly, that "the nitrites are perhaps never required until symptoms of an anginose kind arise."‡

The treatment of valvular disease of the heart by change of climate, regulated activity, gymnastic and other exercises, and by baths, opens up a large subject of which much has been heard in recent years. The methods of Schott, Oertel, and others are well known.

Schott's treatment consists chiefly of "resistance movements" and the use of the aerated brine baths of Nauheim. Oertel's "Terrainkur" consists in regulated walking exercise upon the flat or upon certain graduated inclines. In connection with both lines of treatment dietetic measures play a large part.

* See *ante*, p. 422.

† Practice of Medicine, fourth edition, p. 734.

‡ System of Medicine, vol. v. p. 963.

The fundamental principle which underlies these methods is the advantage of judiciously stimulating the cardiac muscle to increased activity and thereby heightening its growth and nutrition. That exercise of various kinds is, *in suitable cases*, capable of producing such results is certain. That brine baths have a similar effect is not so indubitable. I must confess I read with some incredulity the reports of the rapid diminution of cardiac dilatation under this treatment, and I entertain no doubt that increased expansion of the lungs is the main factor in the alterations in the percussion area of the heart which have been described. I should add, however, that I have made only one visit to Nauheim, and that I have had no adequate opportunity of personally watching the results of the Nauheim methods. The advantage of the Schott and the Oertel methods of treatment would seem to consist not so much in their essential peculiarities as in the fact that they are thoroughly regularized, systematized, and controlled methods. I am inclined to suspect that there is no special magic in "resistance" exercises, that the special form of activity to be prescribed for cardiac patients may well vary from time to time, and that the essential question is rather the selection of cases suitable for active treatment and the due regulation of the kind and amount of such treatment. It may even be plausibly argued that the over-elaboration of rule, law, and supervision may be distinctly injurious by heightening the patient's already excessive morbid subjectivity and preventing the return to a normal psychical state. The psychical condition of cardiac patients is often a point of the greatest importance. Many of these cases are harassed by fears of sudden death, which we know to be, in a large proportion of cases, without adequate foundation. Others, while less fearful, are constantly thinking of their heart, every slight twinge or

nervous flutter—as often as not functional in character and of no consequence—is interpreted as pregnant with grave significance. Such patients are apt—

Propter vitam vivendi perdere causas.

Now, how far the aggregation of such patients in special health resorts, the concentration of thought and emotion on the varied phenomena of cardiac disease, the ever-present spectacle of a certain number of advanced and hopeless cases, may tend to accentuate morbid psychoses or genuine organic symptoms is a grave question.

As to the value of these methods, I will quote the views of three of our most trusted authorities—Broadbent, Douglas Powell, and Strümpell.

On the merits of the Schott treatment, Broadbent writes as follows: "In cases of cardiac dilatation from loss of tone of the heart muscle after influenza, or some depressing disease, it may be of great service, and effect a cure where drugs and other treatment have failed; in many cases of functional and neurotic heart disease, which are very common and difficult to deal with, it may also give satisfactory results. In valvular disease it is, of course, unnecessary when compensation is established and no symptoms present; when compensation has completely broken down it is not advisable, as rest in bed and suitable treatment of other means will be more efficacious. In cases of mitral disease, more especially mitral stenosis, when compensation is just maintained with difficulty, and when the degree of stenosis is such that increased contractile power of the right ventricle induced by digitalis would be useless or harmful, it may be of great service. In aortic disease it is not advisable, owing to the risks of syncopal attacks, though when compensation is breaking down and mitral symptoms are present it may sometimes

yield good results. In adherent pericardium with threatened compensatory failure it may be of service." *

Douglas Powell writes: "In what cases are these exercises to be advised? The employment of the carefully graduated and observed exercises of Schott and Oertel may be regarded as a counsel of perfection to be advised for certain cases only, as a preliminary to the return to that measure of active life of which their heart condition permits, and as serving the purpose of indicating what that measure will be, and by what degree of ordinary exercise it may be arrived at. Resistance exercises are especially adapted for the initial treatment of those flabby, irritable, 'stuffy' hearts, if I may use the term, as applied to cases of fatty infiltration and impaired metabolism, which are met with in people of venous plethora. In cases of chlorosis with dilated heart, after a preliminary week or two of complete rest, the Schott treatment is valuable if combined with a dry bracing climate and some chalybeate. In the first commencing failure of heart in chronic valve lesions, the treatment may be employed combined with a more or less complete cessation from all other exercises, and, similarly, after such cases have been restored up to a certain point by digitalis treatment. Further, certain cases in which from the symptoms and signs we recognize the presence of atheromatous change in the coronary vessels, the treatment may be cautiously tried in combination with much rest. . . . If the treatment be mainly confined to these lines, it is undoubtedly an aid to our therapeutics." †

Strümpell writes: "The employment of baths in heart disease deserves special consideration. Numerous experiences go to prove that they are not only well borne

* Heart Disease, third edition, pp. 105, 106.

† Treatment in Diseases and Disorders of the Heart, pp. 84, 85.

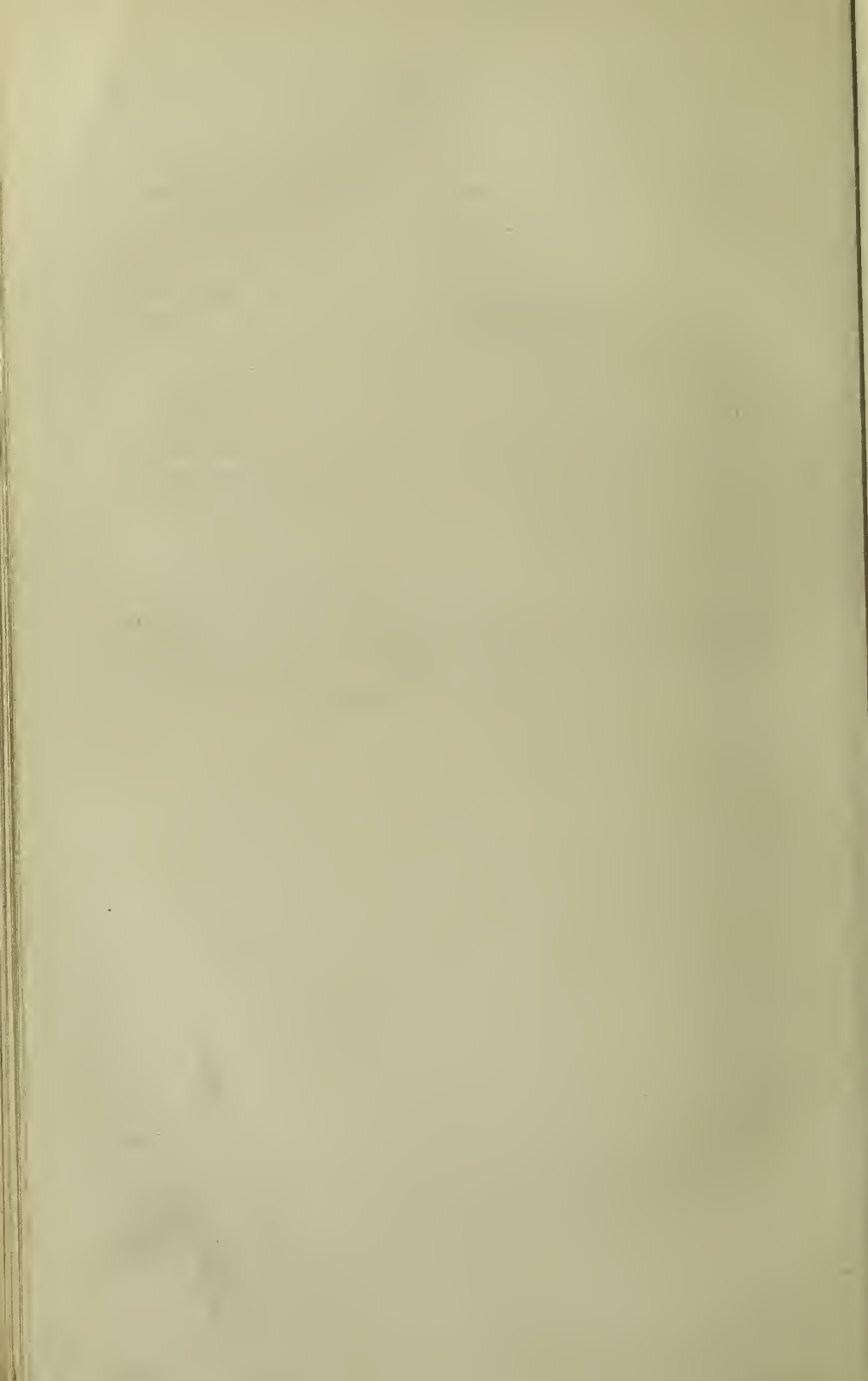
by patients with heart disease, but that they exercise a peculiarly beneficial and invigorating influence upon the action of the heart. In this regard, the greatest reputation is possessed by the warm mud baths. . . . Patients may, also, employ at home either simple, or, still better, salt baths at a temperature of 88° to 90° Fahr. Where there is no marked disturbance of compensation, the methodical employment of gymnastic exercises, the so-called Swedish movements, has a certain value in the treatment of heart disease. Regular muscular movements certainly promote the circulation of the blood, and thus lighten the task of the heart. If they are carried out cautiously and with a proper consideration of the individual case, they are not infrequently beneficial. The best measure for the amount of bodily exercise for patients with heart disease seems to the author to be the subjective sensation of dyspnœa. Any patient who has no special signs of failing compensation may walk at a slow pace until he experiences distinct dyspnœa. As soon as this happens he must stand still and rest. The author regards it as a great mistake to encourage patients to persist in their efforts regardless of difficulty in breathing." *

The concluding remarks of Strümpell point to what is the real question in this connection, viz. not the form of exercise, but the indications for exercise rather than rest. Probably regulated walking in the fresh air on the flat or on certain inclines, according to the indications of the individual case, will achieve most of what may be fairly hoped for from exercise in cardiac cases; but the methodization of the system of Schott, and the amount of supervision and control involved in it, give it, especially with certain types of patient, a distinct advantage. Nevertheless, we see excellent results from the prudent regulation of

* A Text-book of Medicine, English edition, p. 335.

systematic exercise at home without the fuss and ceremony, the questionable environment, and the doubtful psychical influences of the specialized health resort. Dyspnœa is without doubt the most important guide so far as symptoms go. The cardiac patient who can cycle and play golf without any embarrassment of respiration is relieved from the necessity for regular treatment. The patient who has slight degrees of dyspnœa may cautiously try walking on the flat, and judge the value and prudence of such exercises by their effects. In neurotic cases the influence of a special health resort is not free from disadvantage, while a well-regulated holiday in agreeable surroundings and society may do much.

Finally, in the treatment of cardiac disease, the most potent factors are not any departure from simple, recognized, and more or less obvious methods of treatment, but the docility of the patient, the state of his tissues, and the insight and *savoir faire* of the physician.



INDEX OF AUTHORITIES.

-
- | | |
|---|--|
| <p> ALBUTT, 171, 309, 330, 360, 363,
 407, 418, 432
 Anderson, McAll, 175
 Andrew, 291
 Aran, 41
 Aristotle, 200
 Aschoff, 86
 Ashby and Wright, 388
 Auenbrugger, 41
 Aufrecht, 49, 253, 260

 BAAS, 59
 Baccelli, 40, 110
 Baginsky, 387
 Balfour, 262, 299, 360, 370, 382, 426
 Barrs, 85
 Bäumlér, 48
 Beau, 59
 Béclère, 132
 Bennett, Hughes, 260
 Biermer, 51
 Bowditch, 85
 Bradford, 332
 Bramwell, 364
 Brehmer, 171, 180
 Bristowe, 45, 370
 Broadbent, 287, 290, 298, 305, 311,
 315, 319, 326, 331, 333, 344, 345,
 389, 400, 406, 409, 426, 434

 CAPO, Espino y, 132
 Charcot, 5
 Chauveau, 295
 Clark, Andrew, 397
 Clouston, 119
 Corvisart, 41, 280
 Costa, 132
 Coston, 85

 DALLY, Halls, 132
 Denison, C., 211 </p> | <p> Dettweiler, 171, 180
 Dickinson, 354, 355
 Dubville, 85

 EGGER, 214
 Ellis, 47, 94

 FAGGE, Hilton, 260, 261
 Fanning, Burton, 184
 Fiedler, 85
 Finkler, 48
 Flint, 77
 Fox, Wilson, 160, 227
 Foxwell, 385, 386
 Fowler and Godlee, 41, 128, 129,
 130
 Fränkel, 171, 267, 392
 Fräntzel, 315

 GAIRDNER, 281
 Gee, 43, 59, 78, 96, 127, 145, 283,
 291, 296
 Gerhardt, 41, 49, 59
 Gibson, 283, 363
 Gibson and Russell, 287, 325, 336
 Goetsch, 175
 Gordon, W., 205
 Guttmann, 57, 283, 287, 335

 HENOCHE, 414
 Heron, 175
 Hippocrates, 55, 74, 200
 Hoffmann, 269
 Huchard, 328, 329, 361, 363, 365,
 367, 392, 393, 428
 Huggard, W. R., 190, 191

 JACCOUD, 159, 167
 Jürgensen, V., 48, 260

 KELSCH and Vaillard, 85 </p> |
|---|--|

- Kidd, Percy, 160, 165, 171
 Kirchner, 175
 Koch, 86, 175, 176, 177
 Knopf, 171
 Krause, 175
 Kündig, 214

 L'ENNEC, 41, 55, 56, 59, 61, 62, 66,
 69, 73, 74, 75, 76, 79, 228, 280,
 281
 Landouzy, 85
 Lemoine, 85, 86
 Leon, Bonnet, 132, 133
 Leube, 130, 299, 308, 310, 315, 323,
 382
 Little, James, 268, 431

 MACCORMAC, Henry, 180
 Mercier, 214
 Moore, J. W., 253, 260
 Morin, 184

 NAUNYN, 299, 382
 Netter, 86

 O'CARROLL, 291
 Oertel, 432, 433, 435
 Ord, 5
 Osler, 70, 109, 117, 143, 171, 175,
 260, 324, 399, 407, 428, 432

 PHILIP, R. W., 171
 Piorry, 41, 43
 Potain, 312, 316, 368
 Powell, Douglas, 171, 424, 425, 434,
 435
 Prudden, Mitchell, 86

 RANSOME, 171
 Rembold, 175
 Riegel, 374
 Roberts, 101
 Robertson, Argyll, 5

 Rosenbach, 264
 Rotch, 284

 SAHLI, 275, 278, 298, 299, 302, 308,
 335, 337, 369, 373
 Sandwith, 209
 Sansom, 309, 310, 312, 364, 367, 368
 Savill, T. D., 283
 Schlenker, 86
 Schott, 432, 433, 435
 Sée, Germain, 85, 87, 260
 Skoda, 42, 43, 44, 45, 53, 56, 59, 61,
 62, 66, 69, 76, 260, 290
 Smith, Lorrain, 332
 Snow, W. V., 185
 Spengler, 175
 Steel, Graham, 299, 369
 Stephani, 216
 Stokes, 63, 369
 Strümpell, 128, 260, 308, 406, 424,
 435, 436
 Sutton, 240

 TRAUBE, 41, 304
 Trudeau, 171
 Turban, 175, 183, 187, 188, 216, 227,
 228

 VIAULT, 214

 WALSHE, 227, 277, 309, 368, 406
 Walsham, 132
 Weber, Hermann, 171, 183, 213,
 214, 215, 270
 West, 99, 104, 109, 158, 160, 171,
 176, 186
 Wilks, 407
 Williams, C. J. B., 45
 Williams, Theodore, 171, 204, 206
 Wintrich, 41, 44, 54

 YEO, Burney, 221

GENERAL INDEX.

- ACCENT of cardiac sounds, 285
 Accentuation of cardiac sounds, 290
 Accessory sounds in the lungs, 58
 Accident as a cause of disease, 25
 Aconite, in aortic disease, 431
 „ in pneumonia, 263
 Actinomycosis of lungs, diagnosis of, 52, 136
 „ „ distinction of, from phthisis, 136
 Acute onset in phthisis, 159
 Adhesions, pleural, 99
 „ pericardial, 274
 Adirondacks, the, 202
 Adventitious sounds in the chest, 69
 Ægophony, 76
 Africa, South, 210
 Age, influence of, in prognosis of phthisis, 153
 Age, influence of, in prognosis of valvular disease of heart, 413
 Air, of high altitudes, 213
 „ of deserts and inland plains, 208
 „ of the ocean, 202
 „ of marine resorts, 205
 Alar chest, 37
 Allassio, 201
 Albuminuria, in phthisis, 155, 214, 219
 Albuminuria, in pneumonia, 98
 „ in valvular disease of heart, 354
 Alcohol, in heart disease, 420, 431
 „ in phthisis, 194
 „ in pneumonia, 260
 Alcoholism, concealed, rules for detection of, 31
 Algeria, 208
 Aliwal North (Cape Colony), 210
 Alps, climate of, 213
 Alveoli of lung, question of production of sounds in, 70
 Amphoric breathing, 63
 „ hum, 74
 Amusements in heart disease, 420
 Amyloid disease in phthisis, 154, 164
 Anæmia, relation to phthisis, 123, 147
 „ cardiac murmurs in, 307, 379, 383
 Andermatt, 202
 Andes, 202
 Aneurism, of aorta, 234, 320
 „ of ascending aorta, 323
 „ of arch of aorta, 323
 „ of descending aorta, 324
 „ of innominate artery, 323
 Aneurismal phthisis, 323
 Antiseptic treatment of phthisis, 177
 Aorta, atheroma of, 320
 „ aneurism of, 320
 Aortic obstruction, 298, 304, 378, 406
 „ regurgitation, 298, 380, 407, 410, 427
 Aortitis, acute, 319
 Apex beat of heart, displacements of, 277
 Apex beat of heart, importance of, in diagnosis of pulmonary disease, 40
 Arcachon, 201
 Arctic climate, Commandant Peary on, 214
 Areas, cardiac, 285
 Armidale (New South Wales), 212
 Arolla, 202
 Arosa, 202, 215
 Arrhythmia, cardiac, 358
 „ varieties of, 359
 Arsenic, in asthma, 268
 Arteries, physical examination of, 324
 Arthritic hæmoptysis, 226
 Aspiration, in pleural effusion, 102
 Assouan, 209
 Asthma, distinction of, from phthisis, 146

- Asthma, physical signs in, 80
 " treatment of, 265
 Atheroma of aorta, 320
 Auricles, examination of, 278, 279
 Auscultation, of heart, 285
 " of lungs, 55
 " of blood vessels, 321,
 325
 Auscultatory percussion, 46
 Australia, 212

 BACCELLI's sign, 40
 Bacteriology of phthisis, 125
 " of pleural effusion, 86
 " of pneumonia, 257
 Barrel-shaped chest, 37
 Baths, in the treatment of heart dis-
 ease, 432
 Bathurst (New South Wales), 212
 Beaufort West (Cape Colony), 210
 Beechworth (Victoria), 212
 Biarritz, 201
 Biskra (Algeria), 201
 Bloemfontein, 210
 Blood casts, in heart disease, 354
 Blood-letting, in heart disease, 429
 " in pneumonia, 257
 Blood-vessels, physical examination
 of, 318
 Bordighera, 205
 Bournemouth, 206, 270
 Box-tone, 51
 Bradycardia, 329, 373
 Breath sounds, classification of, 59
 Breathing, amphoric, 68
 " bronchial, 65
 " cavernous, 68
 " Cheyne-Stokes, 352
 " harsh, 63
 " puerile, 60
 " tubular, 65
 " vesicular, 59, 62
 Bronchi, foreign body in, 254
 Bronchial râles, 72
 Bronchitis, distinction of, from
 phthisis, 145
 " influence of, upon prog-
 nosis in phthisis, 155
 " physical signs of, 50, 79
 " in pneumonia, 250
 Bronchiectasis, distinction of, from
 phthisis, 140, 236
 " physical signs of, 80
 Broncho-pleural fistula, treatment of,
 264
 Bronchophony, 76
 Broncho-pneumonia, distinction of,
 from phthisis, 141

 Broncho-pneumonia, physical signs
 of, 49, 81
 Bruit d'airain, 75
 Bubbling râle, 72
 Bulging of the chest, 38, 274
 Buxton, 417

 CAFFEINE, in asthma, 269
 " in cardiac disease, 429
 Cairo, 209
 California, Southern, 208
 Canary Islands, 207
 Cannes, 205
 Canterbury Plain (New Zealand), 213
 Capillaries, examination of, 339
 Capillary pulse, 339
 Cardiac areas, 285
 " dilatation, 289
 " dulness, 283
 " hypertrophy, 289
 " impulse, 280
 " murmurs, 293
 " muscle, diseases of, 391
 " sounds, 285
 Cardio-pulmonary murmurs, 312
 Carotid arteries, 324
 Carpathians, 202
 Catania, 208
 Catarrhal onset in phthisis, 159
 Caux, 202
 Cavernous breathing, 68
 " râle, 73
 Cavity, pulmonary, physical signs of,
 53, 71
 Cerebral type of pneumonia, 251
 Chest, alar, 37
 " barrel-shaped, 37
 " examination of, 36
 " pigeon-breasted, 37
 Cheyne-Stokes breathing, 352
 Child-bearing, influence of, upon
 prognosis in phthisis, 153
 Chill, as a cause of disease, 24
 " in pleurisy, 84
 Chlorosis and phthisis, 123
 Chronic pleurisy, treatment of, 109
 Cirrhosis of lungs, physical signs of,
 81
 Clavadel, 202
 Climate, in asthma, 270
 " in phthisis, 197
 " - varieties of, 201
 Cod-liver oil, in phthisis, 194
 Cog-wheel breathing, 64, 127
 Collapse, pulmonary, 67, 139
 Colorado, 211
 Compulsory notification in phthisis,
 174

Conduction of cardiac murmurs, 300
 „ of râles and rhonchi, 73
 Congestion of lungs, physical signs of, 49, 81
 Consecutive onset in phthisis, 160
 Consolidation of lungs, 66
 Convallaria, 429
 Corrigan's pulse, 380
 Cough, in phthisis, 120, 196
 „ in heart disease, 352
 Counter-irritation, in phthisis, 194, 196
 Cracked-pot sound, 45
 Cradock (Cape Colony), 210
 Creosote, in phthisis, 179
 Crepitation, mode of production of, 70
 Cricff, 417
 Criteria of improvement in phthisis, 188
 Cyanosis, in cardiac disease, 275, 347

DAVOS, 202
 Dawlish, 206
 Decubitus, in pleurisy, 98
 „ in pneumonia, 98
 Delirium cordis, 370
 Denver, 211
 Diabetes, relation of, to phthisis, 118
 Diagnosis, principles of, 1
 Diagnostic problems in heart disease, 377
 Diarrhoea, in phthisis, causes of, 164
 „ „ treatment of, 194
 Diastolic murmurs, 313
 „ shock, 282
 Dirotism of pulse, 331
 Diet, in asthma, 268
 „ functional heart disease, 417
 „ organic heart disease, 422
 „ phthisis, 194
 „ pleurisy, 101
 „ pneumonia, 259
 Dieulafoy's aspirator, 105
 Digestive symptoms in heart disease, 353
 Digitalis, in heart disease, 423
 „ in pneumonia, 262
 Dilatation of heart, 277, 278, 279, 280, 283, 288, 292
 Direct method of diagnosis, 6
 Displacements of heart, 40, 277
 Drawbacks to sanatorium treatment of phthisis, 190
 Dropsy, in heart disease, 348
 „ in phthisis, 164

Ductus arteriosus, patency of, 282
 Dulness, cardiac, 283
 „ in phthisis, 53, 125
 „ in pleurisy, 46, 94
 „ in pneumonia, 48
 Dyspepsia, in heart disease, 353
 „ in phthisis, 122, 193
 Dyspnoea, in asthma, 266
 „ in heart disease, 349
 „ in phthisis, 164
 „ in pleurisy, 98
 „ in pneumonia, 98

EARLY diagnosis of pulmonary phthisis, 112
 Egypt, 209
 Ellis's curve, 94
 Embolism of lungs, distinction of, from phthisis, 140
 Embryocardia, 369
 Emphysema, compensatory, in phthisis, 132
 „ physical signs of, 79
 Empyema, complications of, 264
 „ treatment of, 263
 Endemic hæmoptysis, 223
 Epigastric pulsation, 278
 Endocardial murmurs, 295
 Errors of patients in giving history of disease, 24
 Exercise, in heart disease, 421, 432
 „ in phthisis, 182
 Expectoration, in asthma, 266
 „ in phthisis, 125

FAMILY history, in disease, 16, 29, 115, 156, 399
 Fatty degeneration of heart, 391
 Fats, distaste for, in phthisis, 119
 Fibrosis, pulmonary, physical signs of, 50, 81
 „ „ in phthisis, 158
 Fistula in ano, in phthisis, 155
 Flat chest, 38
 Flint's murmur, 315, 380
 Florida, 208
 Fluctuation, pericardial, 282
 „ pleural, 40, 93
 Foramen ovale, persistent, 303
 Foreign body in bronchus, 254
 Formalin, in phthisis, 179
 Fremitus, vocal, 39
 Frémissement cataire, 281
 Friction, pleural, 92
 „ pleuro-pericardial, 294
 Functional disorders of heart, treatment of, 417

- GALLOP rhythm, 368
 Gangrene of lungs, 235
 Gastric type of pneumonia, 251
 Glandular involvements in phthisis, 154
 Glengariff, 206
 Glion, 270
 Goulburn (New South Wales), 212
 Graaf Reinet (Cape Colony), 210
 Grand Canary, 207
 Grasse, 205
 Greytown (Natal), 210
 Griqualand West, 210
 Grindelia robusta, in asthma, 269
 Guaiacol, in phthisis, 179
 Güimar (Canary Islands), 207
 Gurgling sounds in cavities, 73
- HABITS, in heart disease, 399
 „ in phthisis, 180
 Hæmic murmurs, 309
 Hæmophilia, hæmoptysis in, 223
 Hæmoptysis, causes of, 222
 „ dangers of, 231
 „ differential diagnosis of, 232
 „ prognosis in, 231, 236
 „ treatment of, 195, 237
 Hæmorrhagic pleural effusion, 105
 „ onset in phthisis, 159
 Hamman R'Ihra (Algeria), 209
 Harrismith (Orange River Colony), 210
 Harrogate, 417
 Harsh breathing, 63
 Hay asthma, 266
 Headache, in heart disease, 356
 Heart, examination of, 272, 293
 Heart disease, diagnostic problems in, 377
 „ „ prognosis in, 394
 „ „ treatment of, 416
 Heidelberg (Transvaal), 210
 Helouan, 209
 Hepatic type of pneumonia, 251
 Heredity, in heart disease, 16
 „ in phthisis, 115
 Hiccough, in heart disease, 349
 History of disease, interpretation of, 15
 Hydatids of lung, diagnosis of, 52
 Hypopathy in cardiac disease, 417, 432
 Hydrothorax, 349
 Hygiene, of the skin, in phthisis, 194
 „ teaching of, in schools, 174
- Hygienic treatment of phthisis, 180
 Hypertrophy of heart, 274, 277, 280, 283, 284, 289, 292
- ICTERIC tinge, in heart disease, 276
 Ilfracombe, 206
 Indeterminate respiratory murmur, 62
 Infarction, pulmonary, 50, 235
 Influenza, as a cause of phthisis, 118
 Ingravescens murmur, 297
 Inhalations, in phthisis, 178
 „ in asthma, 269
 Innominate artery, aneurism of, 323
 Insanity, relation of, to phthisis, 119
 Insidious onset in phthisis, 159
 Insomnia in heart disease, 357, 419
 Inspection of chest, in heart disease, 273
 „ „ in pulmonary disease, 36
- Integuments, examination of, 37
 Intensification of cardiac sounds, 286
 Intensity of breath sounds, 63
 Intensity, point of maximum, of cardiac murmurs, 298
 Intermittent action of heart, 360
 Intra-tracheal injections in phthisis, 178
 Intravenous injections in phthisis, 178
 Iodide of potash, in asthma, 268
 „ „ in heart disease, 426
- Irregular action of heart, 362
 Irritable aorta, 278
- JUGULAR veins, pulsation in, 337
- KAROO, The (Cape Colony), 210
 Kidney complications, in heart disease, 354
 „ „ in phthisis, 154
- Kimberley, 210
 Knife-grinders' phthisis, 117
- LADYBRAND (Orange Colony), 210
 Lænnec's classification of breath sounds, 61
 Lænnec's classification of râles, 69
 La Laguna (Canary Islands), 207
 Lardaceous disease in phthisis, 154
 Larval type of pneumonia, 253
 Laryngeal phthisis, prognosis in, 154
 „ „ resorts for, 219
 „ „ treatment of, 196
- Las Palmas (Canary Islands), 207

- Les Avants, 202
 Leysin, 202
 Lobelia, in asthma, 268
 Los Angeles, 211
 Lungs, methods of examination of, 35
 ,, congestion of, 81
 ,, fibrosis of, 81
 ,, œdema of, 81
 ,, tuberculosis of, 82
 Luxor, 209

 MACEDON (Victoria), 212
 Madeira, 208
 Malaga, 201
 Malignant disease of lungs, 137, 242
 Maloja, 202
 Manitou Springs (Colorado), 211
 Maranoa (Queensland), 212
 Marine resorts in phthisis, 205
 Marriage, question of, in heart disease, 420
 Matjesfontein (Cape Colony), 210
 Measles, as a cause of phthisis, 118
 Mediastinitis, 335
 Mena House, 209
 Mensuration of chest, 77
 Menthol in phthisis, 178
 Mentone, 205
 Meran, 201
 Mercury, in pulmonary syphilis, 197
 ,, in heart disease, 425
 Metallic tinkling, 74
 Methods of diagnosis, 1
 Micro-organisms, in phthisis, 125
 ,, in pleurisy, 86
 ,, in pneumonia, 257
 Miliary tuberculosis, 52, 168
 Mitral obstruction, 301, 314, 407, 410
 Mitral regurgitation, 301, 305, 406, 411
 Mixed infections in phthisis, 195
 Montana, 202
 Monte Carlo, 205
 Mont Dore, 270
 Monterey (California), 208
 Montreux, 201
 Morphia, in heart disease, 431
 ,, in pneumonia, 259
 Mountain climates, 213
 Mount Victoria (New South Wales), 212
 Movements of chest, 38
 Murmurs, cardiac, 293
 Myocarditis, 328, 367, 391

 NAPIER (New Zealand), 213
 Naso-pharynx, relation of, to asthma, 266

 Natal, 210
 Nauheim treatment of heart disease, 432
 Nervous symptoms in heart disease, 355
 Neurotic dyspepsia, distinction of, from phthisis, 147
 New South Wales, 212
 New Zealand, 213
 Nice, 205
 Night-sweating in phthisis, treatment of, 194
 Nile voyage, the, in phthisis, 209
 Nitrites in heart disease, 432
 Nux vomica, in asthma, 269
 ,, in phthisis, 194

 OCCUPATION, question of, in heart disease, 415, 420
 ,, question of, in phthisis, 117
 Ocean voyage, the, 202
 ,, cases suitable for, 203
 Œdema, in heart disease, 275, 348
 ,, of lungs, in heart disease, 350
 ,, physical signs of, 81
 Oertel treatment of heart disease, 432
 Onset, modes of, in phthisis, 119, 159
 ,, prognostic indications of, in phthisis, 159
 Orange River Colony, 210
 Orotava, 207
 Oxygen, in pneumonia, 262

 PAIN, interpretation of, 32
 ,, in heart disease, 343
 ,, in phthisis, 123, 196
 ,, in pleurisy, 97
 ,, in pneumonia, 97
 Palermo, 208
 Pallor, in heart disease, 274, 347
 Palpation, 39, 279, 321
 Palpitation, 341
 Paracentesis thoracis, 102
 Paraldehyde, in cardiac insomnia, 431
 Pasadena (California), 211
 Pau, 201
 Percussion, 41, 283
 Pericardial effusion, 274
 ,, fluctuation, 282
 ,, friction, 282
 Personal history in disease, 18, 115
 Phonendoscope, the, 58, 273
 Phthisis, conditions which simulate, 135

- Phthisis, early diagnosis of, 112
 " physical signs in, 82, 125
 " prognosis in, 151
 " treatment of, 171, 192
 Pigeon-breasted chest, 37
 Pleurisy, 46, 67, 84, 143, 155
 " influence of, upon prognosis
 in phthisis, 155
 " relation of, to tuberculosis,
 85
 Pleximeters, 41
 Pneumonia, atypical, 248
 " diagnosis of, 80, 97
 " physical signs of, 48
 " treatment of, 256
 Pneumothorax, 83, 154
 Posture of patient during examina-
 tion, 35, 273
 Posture of patient, influence of, upon
 cardiac murmurs, 316
 Post-tussive suction, 75
 Pre-systolic murmurs, 314
 Prognosis, in phthisis, 151
 " in pleurisy, 100
 " in valvular disease of
 heart, 394
 Pulmonary obstruction, 299
 " regurgitation, 299
 " second sound, accentua-
 tion of, 290
 Pulsation, abnormal, in cardiac area,
 276
 " capillary, 339
 " venous, 336
 Pulse, examination of, 326
 " frequency of, 327
 " intermittent, 360
 " irregular, 362
 " phthisical, 123
 " rhythm of, 358
 " tension of, 330
 " volume of, 330
 Pulsus bigeminus, 367
 Pyrexia, in phthisis, 122, 154, 163,
 195
 " in pleurisy, 104
 " in pneumonia, 250

 QUALITY of breath sounds, 61
 " of cardiac murmurs, 296
 Queensland, 212
 Queenstown, 206
 Quinine, in pneumonia, 263

 RADIOSCOPY, in the diagnosis of pul-
 monary disease, 132
 Râles, classification of, 70

 Rapallo, 205
 Rarer forms of pulmonary disease,
 242
 Recession of the chest, 39
 Reduplication of the cardiac sounds,
 367
 Resistance exercises in cardiac dis-
 ease, 432
 " sense of, in percussion,
 46
 Resonance, amphoric, 45
 " Skodaic, 44
 " vocal, 75
 Respiration soufflante, 61
 Respiratory gymnastics, in pleurisy,
 109
 Response to treatment, in heart dis-
 ease, 401
 " " in phthisis,
 155
 Rest, in heart disease, 421
 " in phthisis, 182
 Retraction of the chest, 274
 Rheumatism, in heart disease, 405
 " in pleurisy, 85
 Rhonchal fremitus, 40
 Rhonchi, 69
 Rhythm, cardiac, disorders of the,
 358
 " respiratory, 64
 " of cardiac murmurs, 302
 Rhythme couplé du cœur, 366
 Riviera, The, 205
 Rostrevor, 206

 SALCOMBE, 206
 Sanatorium treatment of phthisis, 181
 " " " re-
 sults of, 183
 San Diego (California), 208
 San Remo, 205
 Santa Barbara (California), 208
 Santa Cruz (Canary Islands), 207
 Schott treatment of heart disease,
 432
 Sea voyage, in phthisis, 202
 Serial method of diagnosis, 3
 Sex, influence of, in prognosis of
 heart disease, 414
 Sex, influence of, in prognosis of
 phthisis, 153
 Sicily, 208
 Sidmouth, 206
 Skoda's classification of breath
 sounds, 61
 " " of percussion
 sounds, 43
 Skodaic resonance, 44

Sounds of heart, 285
 Specific treatment of phthisis, 175
 Sputum, significance of, in phthisis, 124
 Staccato percussion, 42
 Stethoscopes, 57
 Strophanthus, in pneumonia, 262
 " in heart disease, 429
 Strychnine, in pneumonia, 262
 " in heart disease, 426
 Study of cardiac symptoms, the, 340
 St. Leonards, 206
 St. Moritz, 215
 Succussion splash, 74
 Symptoms, of heart disease, 340
 " of pleurisy, 97
 " of pneumonia, 97
 Syncope, in heart disease, 355
 Syphilis, pulmonary, 138
 " relation of, to phthisis, 119
 Systolic murmurs, 302

TACHYCARDIA, 370
 Tangier, 208
 Taormina (Sicily), 208
 Tapping, in pleural effusion, 102
 Tasmania, 212
 Temperature, in phthisis, 154
 Tension of pulse, 330
 Terrainkur, Oertel's, 432
 Therapeutic problems in pulmonary disease, 256
 Thrills, cardiac, 280
 Timbre of cardiac sounds, 291
 Tongue, state of, in phthisis, 193
 Toowoomba (Queensland), 212
 Torquay, 206
 Tracheal tugging, 322
 Transvaal, the, 210
 Treatment, antiseptic, of phthisis, 177
 " of asthma, 265
 " of broncho-pleural fistula, 264
 " climatic, of phthisis, 197
 " of functional disorders of heart, 417
 " of hæmoptysis, 237
 " hygienic, of phthisis, 180
 " Oertel's, of heart disease, 432
 " of organic disease of heart, 419
 " of pleurisy, 101
 " of pneumonia, 256
 " preventive, of phthisis, 172
 " Schott's, of heart disease, 432

Treatment, specific, of phthisis, 175
 " symptomatic, of phthisis, 192
 Tricuspid obstruction, 299, 307, 315
 " regurgitation, 299, 307, 389
 Tuberculin in phthisis, 175
 Tuberculosis of lungs. *See* Phthisis.
 Tussive fremitus, 40
 Tympanitic percussion sound, 44
 Types of patient in phthisis, 158, 165, 216
 Typhoid fever, as a cause of phthisis, 118

ULCERATION of bowels, 155, 164, 194
 " of bronchi, 222, 235
 " of larynx, 154, 196, 219
 " of mouth, 193
 Urine, condition of, in heart disease, 354
 " " in phthisis, 155

VARIATIONS of the physical signs in phthisis, 131
 " in intensity of cardiac sounds, 286
 Vascular degeneration, 398, 405
 Vaso-dilators in heart disease, 432
 Veins, examination of, 334
 Venesection, in heart disease, 429
 " in pneumonia, 257
 Venous hum, 338
 " pulse, 336
 " thrill, 339
 Ventnor, 206
 Ventricle, left, dilatation of, 277, 280, 284, 292
 " " hypertrophy of, 277, 280, 284, 289, 292
 " right, dilatation of, 277, 284
 " " hypertrophy of, 277, 284
 Vertigo, in heart disease, 356
 Vocal fremitus, 39
 " resonance, 75
 Vomiting, in heart disease, 353
 Voyage, sea, in phthisis, 202

WEAK breathing, 63
 Weight, significance of, in phthisis, 116, 163, 189
 Weissenburg, 270
 Whooping-cough, as a cause of phthisis, 118
 Wiesen, 202
 Wintrich's sign, 44, 54
 Woodhall Spa, 270

PRINTED BY
WILLIAM CLOWES AND SONS, LIMITED,
LONDON AND BECCLES.

